Pikul K.V., Ilchenko V.I., Prylutzkiy R. Yu.

Manual for foreigners

students of higher medical education

INTENSIVE CARE FOR THE CHILDHOOD INFECTIOUS DISEASES CLINICS

Міністерство охорони здоров'я України
Українська медична стоматологічна академія

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Poltava 2020

Ministry of Healthcare of Ukraine
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Навчально-методичний посібник підготовлений співробітниками кафедри ендокринології з дитячими інфекційними хворобами (зав. кафедри проф. Л.Є. Бобирьова) Української медичної стоматологічної академії, доц., к.мед.н. К.В.Пікуль, доц., к.мед.н. В.І.Ільченко, к.мед.н. К.Ю.Прилуцьким.

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List of abbreviations

ABB - acid-base balance
ACV is hepatitis C virus
AII - acute intestinal infection
AR - agglutination reaction
AS - acetonemic syndrome
ASLT - an acute stenotic laryngotracheitis
BBB - blood-brain barrier
BES - brain edema-swelling
BR - breathing rate
CBR - complement-binding reaction
CHB - chronic hepatitis B.
CIP - complex immunoglobulin preparation
CVP - the central venous pressure
EE - enteropathogenic Escherichia ETE -enterotoxigenic Escherichia
EI - enzyme immunoassay
HAIR - hemad absorption inhibition reaction
HAV - hepatitis A virus
HBV - hepatitis B virus
HDV - hepatitis D virus
HIR - hemagglutination inhibition reaction
IAR - immunofluorescence analysis reaction
IFR - immunofluorescence reaction
ITS - an infectious-toxic shock
NR - neutralization reaction
OR - oral rehydration
PHR - passive hemagglutination reaction
PHR- passive hemagglutination reaction
PR - precipitation reaction
RIA-radioimmunoassay
VH - viral hepatitis

Навчально-методичний посібник складений згідно з типовим навчальним планом та програмою для підготовки іноземних студентів медичних закладів вищої освіти для циклу «Дитячі інфекційні хвороби». Цей навчально-методичний посібник написаний англійською мовою і присвячений питанням етіології, епідеміології, клініки та диференційної діагностики найбільш частих дитячих інфекційних хвороб, наведені алгоритми діагностики та терапії, згідно з протоколами лікування, що затверджені МОЗ України. Назви розділів автори підібрали у відповідності з тематичним планом для підготовки іноземних студентів медичних закладів вищої освіти. Посібник містить запитання для самоконтролю, тести, задачі та розбір історій хвороб дітей, що лікувались у дитячому відділенні ПОКІЛ для більш глибокого засвоєння матеріалу.

The teaching manual is compiled according to a typical curriculum and a program for the preparation of foreign students of medical institutions of higher education for the cycle "Children's Infectious Diseases". This teaching manual is written in English and is devoted to the issues of etiology, epidemiology, clinics and differential diagnostics of the most common infectious diseases of children, diagnostic algorithms and therapies are given in accordance with the treatment protocols approved by the Ministry of Health of Ukraine. The titles of the sections were chosen by the authors in accordance withthe thematic plan for thepreparation of foreign students of medical institutions of highereducation. The manual contains questions for self-control, tests, tasks and analysis of children's diseaserecords that were treated at the children's department for a deeper assimilation of the material.

Manual

Infectious diseases have always been dangerous, which indicated on their mass prevalence, rapid spread and high mortality rates, especially in childhood. Many infections can lead to the development of urgent conditions, which requires intensive care, taking into account the etiological factor of the pathological process.

This manual is devoted to the issues of etiology, epidemiology, clinics and differential diagnosis of the most common emergencies among pediatric infectious diseases, and provides algorithms for emergency care and intensive care. The manual contains questions, tests, self-monitoring tasks that will help students and interns learn material better.

We hope that the manual we have written will be useful in the daily practical work of pediatricians, pediatric infectious diseases doctors and family physicians.

The authors gratefully acknowledge all wishes and critical remarks.

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HYPERTHERMIC SYNDROME AND FEVER IN CHILDREN

Depending on the mechanism of development of fever, there are: hyperthermic syndrome and feverish condition.

Hyperthermic syndrome is a prolonged (more than three hours) increase in body temperature above 38.5-40°C, fever, tachycardia, respiratory arrhythmia, drop in blood pressure, hallucinations (visual), clonic-tonic convulsions, loss of consciousness.

The reason: the result of a disturbance of heat balance due to insufficient efficiency of mechanisms of thermoregulation or disruption of their function - so-called hyperthermic reactions - they are inadvisable for the body; hardly relieved by antipyretics, effective overall body cooling. Hyperthermic syndrome is considered as an initiating phase of infectious-toxic shock.

Under the influence of pathogenic factors in the condition of toxicosis the catecholamines release, activation of phosphorylation in the liver, muscles, which enhances heat production and causes spasm of peripheral vessels and leads to a decrease in heat transfer. Acidosis, hypercapnia, hypoxia, disorders of central mechanisms of thermoregulation are observed.

The fever is the result of exposure to pyrogens (bacterial or viral along with the inflammation etc.) that shift the "set point" of body temperature to a higher level due to the activation of thermoregulatory mechanisms. Feverish conditions are biological protective reactions that are more likely to be positive; it is characterized by the effectiveness of antipyretics while the overall cooling of the body has no effect.

The leading mechanism in the genesis of the syndrome - fever are interleukins, tumor necrosis factor etc., which are produced by peripheral mononuclear phagocytes, which are activated under the influence of pyrogen and act on the center of thermoregulation in the hypothalamus. It has been proved that the pyrogenic properties of endotoxin-gram-negative pathogens are the parts of the cell membrane lipopolysaccharides.

Infectious or non-infectious inflammatory process is accompanied by activation of phagocytosis and increased synthesis of phagocytes of biologically active substance, which leads to an increase in body temperature, so-called endogenous or leukocyte pyrogen. Subsequently, it was found that the leukocyte pyrogen is not homogeneous, but it is a group of proteins, among which two active polypeptides were isolated, which is designated as interleukin-1 (IL-1). IL-1 is considered to be a major mediator in the mechanism of fever genesis and of other processes of acute inflammation phase. It stimulates the secretion of prostaglandins, amyloids A and R, S-reactive protein, haptoglobin, α1-antitrypsin and ceruloplasmin.IL-1 action triggers the production of interleukin-2 by T-lymphocytes and it increases the expression of cellular Ig-receptors. In addition, there is increased proliferation of B lymphocytes and stimulation of antibody secretion. Under normal conditions, IL-1 does not penetrate the blood-brain barrier. However, in the case of impaired immune homeostasis under the influence of the infectious or non-infectious inflammation, IL-1 reaches the preoptic region of the anterior part of the hypothalamus and interacts with the neurons receptors of the center of thermoregulation. This activates the cyclooxygenase, which leads to an increase in the intracellular level of cyclic adenosine-3', - 5'-monophosphate. Increasing the concentration of cAMP promotes intracellular accumulation of Ca2 + ions, changes in cell Na / Ca ratio and rearrangements of activity of the centers of heat production and heat transfer with increase in thermal energy and decrease in heat transfer. Increased heat production is achieved by enhancing the metabolic processes of thermogenesis. At the same time, the vessels of the skin and subcutaneous tissue are narrowed, the velocity of the peripheral vascular blood flow decreases, which leads to a decrease in heat transfer. A new, higher level of temperature homeostasis is established which in turn leads to the increase in body temperature.

A typical circadian rhythm of temperature curve variations is formed in children under 2 years of age. The highest values are fixed from 17.00 to 19.00, the lowest - from 2.00 to 6.00, the permissible daily fluctuations are from 0.10

C to 1.30 C. The most likely causes of fever are the following: lished, leading to an increase in body temperature.

- heat stroke, poisoning, toxic poisoning;
- circadian rhythms, physical activity, food intake, stress, ovulation in women;
- non-infectious diseases - autoimmune hepatitis, thyroiditis, thyrotoxicosis, hypercorticism, hypothalamic syndrome in adolescents, neoplastic processes, hematological diseases, manifestation of connective tissue diseases;
- infectious agents - tuberculosis bacilli, HIV, streptococcus, staphylococcus, meningococcus, pneumococcus, enterococcus, hemophilic bacilli, listeria, chlamydia, mycoplasma, DNA viruses, in particular herpes viruses and others.

Types of temperature curves are:

- intermittent - characteristic of infectious diseases, as well as for systemic diseases, autoimmune nosologies, tuberculosis, neoplasms. Sometimes the intermittent temperature curve is combined with the bradycardia observed in such nosologies as - viral myocarditis, rheumatoid arthritis, Lyme disease, infectious myocarditis, atrioventricular blockade;
- remittent - uncontrolled decrease in temperature several times a day is more characteristic of acute bacteremia;
- permanent - characteristic of typhoid fever, miliary tuberculosis, brucellosis, leptospirosis;
- reverse - observed in malaria, Sadoku disease, borreliosis. It should also be noted two more fever variants – septic one and Ebstein (lymphogranulomatosis, histiocytosis).

In hyperthermia, infants and young children often experience febrile seizures. In such cases, it is always necessary to exclude CNS pathology. It can be either an encephalitic reaction, that is, a nonspecific manifestation of the nervous system to various negative factors (toxin, microbe), or seizures in

inflammatory organic processes in the CNS, or a separate nosology (epilepsy, etc.). According to the literature in recent years, the severity of the process is given by herpes viruses, especially type 6, which is an agent of seizures syndrome. Therefore, children with a history of seizures should consult a pediatric neurologist and an infectious disease specialist.

Establishing a preliminary diagnosis: Fever of unknown genesis, leads to delayed treatment and causes loss of confidence in the patient, so it is necessary to establish the cause as soon as possible and to appoint in time the following examination and therapy: **first stage -** clinical systematic examination, general clinical and biochemical methods studies, feces analysis, examination of the immunogram, bacteriological and virological examination of mucus, feces, urine, eye discharge, blood test for sterility, Mantoux, lungs, thymus X-ray, abdominal renal, cardiac, cerebral ultrasonography, **the second stage** - repeated bacteriological and virological examination of mucus, feces, urine, eye secretions, determination of markers of hepatitis, infectious mononucleosis, determination of intracellular pathogens - chlamydia, mycoplasma, DNA viruses by EI and PCR. Brain MRI is recommended**; the third stage** involves invasive methods - puncture of the sternal, spinal and lymph nodes, hepatobiopsy, consultation of the surgeon, ophthalmologist with examination of the ocular fundus, hematologist, immunologist, neurologist.

Why can fever persist for a long time as a leading syndrome? According to the literature, we respond as follows: stress, secondary immunodeficiency, reduction of blood proteins leads to impaired hemodynamics and development of dysbiosis, and as a consequence the anaerobic flora multiplication, increased concentration of toxins, foreign antigens, polysubstrates in the small intestine and emergence of endogenous intoxication. One of the mistakes of outpatient doctors is that they do not see signs of toxicosis in the early stages of diagnosis. Toxicosis is manifested by changes in hemodynamics, nervous system and disorders of water-electrolyte balance. The first sign of toxicosis is pale skin, dark circles under the eyes,

cyanosis, marbling; the second sign is a CNS disorder (excitability, followed by the flaccidity, which is characteristic of bacteria), convulsions; the third sign is dry mucous membranes and face.

Particular attention should be paid to the clinical features of the conformityof heat transfer processes to the increased level of heat production, because, depending on the individual characteristics and background of fever, even with the same level of hyperthermia, children may have different course. Thus, if the body temperature increases, the heat transfer corresponds to the heat production, it indicates an adequate course of fever. Clinically, this is manifested by the normal behavior and well-being of the child, pink or moderately hyperemic skin, moist, warm to the touch ("Pink fever"). Such course of fever is considered as prognostically favorable. If, as the body temperature rises, the heat transfer is substantially less than the heat production, then fever may have an inadequate course. Clinically, it is noted by the disorders of the state and well-being of the child, pale skin remained, acrocyanosis, cold feet and palms ("pale fever"). These clinical manifestations indicate a pathological fever, are prognostically unfavorable, and are a direct indication for emergency care.

Children at risk of developing complications in fever are:

- Under 2 months of age at temperatures above 38 ° C;
- History of febrile seizures;
- With CNS diseases;
- With chronic pathology of the circulatory system organs, respiratory system organs;
- With hereditary metabolic diseases.

Antipyretic therapy for children should be carried out at axillary body temperature not lower than 38 ° C. However, if the child on the background of fever, regardless of the severity of its manifestation, worsening which is accompanied by the myalgia, impaired skin, pallor and other manifestations of toxicosis, antipyretic therapy should be prescribed immediately. Children at risk for the development of complications require the administration of antipyretic

drugs, even at subfebrile temperature. If we do not adhere to these postulates, we can erase the clinical picture to correctly make the clinical diagnosis and break the protective immune cascade of the child's body.

The drugs of choice for fever in children are paracetamol and ibuprofen. It is considered that ibuprofen (Nurofen for children) can be used as starting therapy in cases when the administration of paracetamol is contraindicated or ineffective, in addition, the use of the drug in children from 3 months of age is allowed with some caution. Recommended single doses: ibuprofen - 5 - 10mg/kg (Nurofen 5ml suspension contains 100mg ibuprofen), paracetamol- 10-15mg/kg. Repeated use of antipyretic drugs is possible not earlier than 4-5 hours after the first administration 2-3 times a day. Acetylsalicylic acid and nimesulide are not used for children. Prescription of antipyretic drugs in a course dose at fever is forbidden. If the antipyretics are ineffective, the patient's condition worsens, we consider this situation as a hyperthermic reaction. Tactics: to increase the heat transfer and decrease the heat production: nitroglycerin under the tongue (children under 1 year - ¼ - ⅓; from 1 year to 3 years

• ⅓ - ½; older than 3 years - ½ -1 tablet); droperidol solution 0.25% single dose of 0.05-0.1ml/kg - intravenously slowly for 10% glucose solution, it can be re-administered in 4-8 hours; a pipolpene solution 2.5% (dimedrol 1%) single dose of 0.05 (0.1)ml/kg - intravenously slowly; saline solution; it can be re-administered in 6-8 hours; aminazine solution 2.5% single dose of 0.1-0.2ml/kg intravenously slowly on 10% glucose solution; it can be re-administered in 8-12 hours; diazepam (Relanium, Seduxen) 0.5% solution single dose of 0.1 ml / kg - intravenously slowly; it can be re-administered in 8 hours; or sodium oxybutyrate 20% solution of 0.25-0.5-0.75 ml / kg single dose intravenously slowly; re-enter after 6 hours; hydrocortisone (prednisolone) single dose of 4-5 (2-2.5)mg/kg - intravenously (fluidly or dropwise) in 10% glucose solution, with an interval of 6-8 hours. You can apply lytic mixture - 2.5% aminazine solution 1ml + 2.5% pipolphene solution - 1ml + 0.25% novocaine solution -

8ml; take 0,1-0,15ml/kg is allowed if necessary up to 4-6 times a day. When pink fever we use physical methods of cooling: blowing with a stream of cold air (fan), rubbing the skin with alcohol in half with water, ether, placing something cold on the head and main vessels (inguinal areas), cold wrapping, gastric lavage, intestinal shower (siphon enema) - carrying out these methods, it is necessary to be aware of the possibility of "cold" cardiac arrest! The whole complex of the above mentioned therapy should be carried out in combination with etiotropic treatment and other methods of correction of disturbed homeostasis (normalization of water-electrolyte balance).

Control questions:

1.What are the central mechanisms of thermoregulation?

2.What urgent measures are used for white fever?

3. What urgent measures are used for "pink" fever?

MENINGITIS AND MENINGOCOCCEMIA

Meningococcal infection is an important healthcare problem. It can be realted to the high incidence of mortality and morbidity. About 500,000 cases are recorded every year around the world, about 50,000 of which are fatal. Every year in Ukraine, children are diagnosed with meningitis, in particular of meningococcal etiology, from 800 to 1200 cases, about 100 of which lead to death.

Meningitis is a group of infectious diseases characterized by inflammation of the membranes of the brain and spinal cord. In the structure of neuroinfections, meningitis occupies the second place and make up approximately 23%, 66% of which are in children. In 12.3% of all meningitis, bacteria are the causative agent; in 53.7% - viruses, in 34% - etiology remains unknown for various reasons. The most common causes of bacterial meningitis depending on the age are following: in infants - E. coli, group B streptococci, L. monocytogenes, S. pneumoniae; in children - N. meningitidis, S. pneumoniae, H. influenzae; in adults - S. pneumoniae, N. meningitidis, gram-negative bacilli, Listeria strains. Among meningitis of viral etiology, the most common are enteroviruses and herpes viruses.

In newborns, the origin of meningitis is influenced by perinatal pathology: prematurity, infection in the mother, adverse course of pregnancy or its outcomes. There are primary and secondary meningitis. According to the localization of the process there are diffuse and limited ones, on the basis of the brain - basal or on a convex surface - convex. Depending on the course there distinguish dynamic form, acute, subacute and chronic meningitis, and according to the degree of course - mild, moderate, severe and extremely severe form.

The entrance gate are the upper respiratory tract or digestive system, then the infection reaches the brain membranes hematogenously. A contact way of spreading is also possible.

Table 1

Classification of meningitis

№	Purulent meningitis	Serous meningitis
1.	Bacterial: meningococcal, pneumococcal, Niv-meningitis, staphylococcal, streptococcal, escherichiotic, salmonellosis, enterococcal, proteic, klebsiellosis, pseudomic, anthrax, leptospirosis, listeriosis, mycoplasma, chlamydial and other borreliosis.	Bacterial: tuberculosis, ornithosis, brucellosis, syphilitic, listeriosis, leptospirosis.
2.	Fungal: candidiasis, aspergillosis.	Fungal: blastomycosis, cryptococcal.
3.	Caused by the simplest: amoebic.	Caused by the the simplest: toxoplasmic.
4.		Viral: herpetic, enterovirus, mumps, influenza, parainfluenza, adenoviral, PC-viral, poliovirus, measles, rubella, chickenpox, acute lymphocytic choriomeningitis (Amstron's disease), bocavirus, metapneumovirus.

The main pathogenetic links of meningitis are inflammation and swelling of the meninges, dyscirculation in cerebral vessels, hypersecretion of cerebrospinal fluid and delay of its resorption, which leads to increased intracranial pressure. The pathological process also involves the cranial nerves, ventricles, vascular plexuses. Exudate is contained in the fissures of the brain. In purulent meningitis, the subarachnoid space is filled with fyrinous-purulent exudate, which can be affected by the phagocytosis with macrophages during

the first 3 days under the condition of proper treatment, and reparative changes occur within 2-4 weeks. On 5-6 days, a "pus cap" is formed, covering the hemispheres of the brain. During the organization of pus, adhesions occur in the form of enclosed cysts, obstructed Mozhandi openings, disturbed liquor outflow, which leads to increased intracranial pressure and the development of hydrocephalus. There occur thromboses of blood vessels and hemorrhages.

Meningococcemia is a meningococcal sepsis that develops against the background of a acute sensitization of the body caused by N. Meningitidis and its endotoxin.

Clinical diagnostical criteria of meningococcemia:

- sudden, acute onset, with fever up to 38-40.0C;
- pronounced intoxication syndrome: general weakness, headache, muscle pain, pallor of the skin; Most patients have a spotty-papular rash on the skin without localization within a few hours. After a few hours, on the skin of the buttocks, hips, shin, lower part of the body forms hemorrhagic elements of the rash in size from 1-2mm to several centimeters. Subsequently, necrosis is formed in the center of the largest elements of the rash;
- hemorrhages in the sclera, mucous membranes of the throat, nasal, gastric bleeding may be observed;
- in dynamic form - rapidly developing the manifestations of infectious-toxic shock, hypostatic bluish spots are formed on the body.

The clinical picture of meningitis was described as early as the 7th century by Paul Eginsky. However, one of the most likely signs of meningitis was reported in 1884 by Obukhiv hospital doctor V. M. Kernig, who proved that "a symptom of knee joints contracture" is an early objective manifestation of inflammation of the meninges.

Most often, the onset is acute, rapidly increasing symptoms, fever. In the clinical picture there are three main syndromes:

• hypertensive, which is composed of the following symptoms - headache, vomiting, unrelated to food, blasting of the frontal fontanel, skin hyperasthesia;

• meningeal is a tonic muscle tension or muscle contracture (Bruzinski, Kernig). The cause of this syndrome is irritation by the inflammatory process of III-IV ventricles of the brain, spinal nerve roots and their reflex protection. In children younger than 3 months, diagnostic errors are possible due to similarity with physiological reflexes, so we should check for a symptom of Lesage, and from 6 months it is recommended to notice the symptom of "landing" (with meningitis, the child will not sit);

• liqourological - cytosis, pressure increase up to 200-300 mm water column, protein. Sugar and chlorides are either standard or reduced, depending on the type of meningitis. **Indications for** lumbal puncture: fever, headache, repeated vomiting, positive meningeal symptoms.

Clonic-tonic convulsions occur before or after meningeal syndrome, or accompany it. Seizures are prone to recurrence (the younger the baby, the more frequent the recurrence), may occur according to the type of epileptic status. The cranial nerves are often affected by the type of toxic or infiltrative neuritis. Most often 3, 6, 7, 12 pairs of cranial nerves. involved are affected. Muscle tone is usually reduced and tendon reflexes are increased. There is an anisoreflexion sometimes (on one side the reflexes is higher than on the other). In severe intoxication, reflexes may be absent due to the toxic effect on the reflex arc. Often there are foot clonus and pathological reflexes Babinsky, Rossolimo. Paralysis and paresis are rare only when encephalitis is associated.

Intoxication are a big part of it, which creates a background and often causes circulatory, water-salt and hormonal disorders. In bacterial meningitis, the phenomena of toxicosis will be significantly pronounced, which can cause infectious-toxic shock.

Table 2

Composition of the liqour in children

№	Indicator	Normative
1.	color	colorless, transparent
2.	pressure	100-150mm water column
3.	cytosis (neutrophils are absent, lymphocytes are single)	in newborns normal 25-20 lymphocytes in 1 mkl; in children from 3 months to 1 year - 12-15 lymphocytes in 1 mlk; older children - 1-5 lymphocytes. in 1mkl.
4.	protein	0,1 – 0,3g/l
5.	chlorides	7-7,53g/l
6.	glucose	2,5-4,4mmol/l
7.	sediment tests (Pandit, Nonre,Apelta)	negative
8.	the presence of bacteria (bacteria)	missing

Diagnosis of meningitis includes the following scheme:

• 1.General blood test - neutrophilic leukocytosis with left shift, lymphocytosis, increased ESR;

• 2. Liquor analysis - neutrophilic pleocytosis, increased protein levels, reduced sugars and chlorides;

• 3. Bacterioscopic examination of liquor sediment and blood smears - "thick drop";

• 4.Bacteriological inoculations for selective nutrient media - liqour, blood, nasal mucosa for isolation of pathogen;

• 5.Virological examination of blood, cerebrospinal fluid;

• 6.Serological methods (latex agglutination reaction (LAR), counter-immunoelectrophoresis (CIPH) for determining the pathogen antigen;

• 7. PCR;

• 8.Computer tomography to exclude the volume process;

• Examination of an ophthalmologist with an evaluation of the ocular fundus.

Table 3

Clinical criteria for shock in meningitis.

Clinical	Phase 1	Phase 2	Phase 3
Signs	compensated shock	subcompensated shock	decompensated shock
Complaints	headache, muscle aches, joints ache	sharp weakness	Feeling of cold, lack of air
Body T	38C-39C more than 12 hours	normal or subfebrile	reduced or normal
Skin T	cooling of distal extremities	the skin is cold	the skin is cold
Skin color	pallor, cyanosis of lips and nails, "vasomotor changes", skin marbling "	sharp pallor, acrocyanosis, marbling of the skin	sharp pallor, total cyanosis of skin and mucous membranes, appearance of hypostases
Rash	hemorrhagic, small, plentiful, quickly pours Localization in typical sites	large, abundant with foci of necrosis. The appearance of rash elements on the upper half of the trunk, hands	large plentiful with necrosis, mainly on the trunk, face, neck
Neuro-psychic status	Consciousness preserved, excitability, less lethargy, euphoria, anxiety, hyperesthesia	consciousness is confused, sopor condition, inhibition, adynamia	Sopor, a coma
Breath	the frequency is moderately increased	the frequency is dramatically increased	tachypone
Pulse	satisfactory, rhythmic	weak, rhythmic	filamentous, arrhythmia possible, not determined
Heart rate	tachycardia (heart rate more than normal by 20-30%), heart tones are weakened	sharp tachycardia (heart rate exceeds the norm by 50% - 60%)	severe tachycardia (heart rate exceeds the norm by 80%) or bradycardia
BP	normal or elevated	reduced to 70-50% of the age limit	below 50% of age, undetermined
Diuresis	remains	oliguria	oliguria or anuria
CVP	normal	reduced	increased

An algorithm for providing medical care for the children with meningococcemia

- Provision of venous access.
- Ensuring airway patency.
- Oxygen therapy with 100% oxygen. If signs of phase II-III shock, cerebral edema, convulsions - after pre-medication with 0.1% atropine sulfate solution at a dose of 0.1ml/ year of life (no more than 0.5ml), intravenously, with ketamine at a dose of 5mg/kg intravenously tracheal intubation is performed intravenously and the patient is transferred to mechanical ventilation.
- Infusion therapy with saline, colloidal solutions for stabilization of BCC.
- Intravenous administration of ceftriaxone. Benzylpenicillin may be used in mild and moderate forms of meningococcemia.
- Glucocorticosteroids intravenously (prednisolone, hydrocortisone) at a dose of 5mg/kg - with TTS of Ist degree, 10mg/kg - with IInd degree, 15-20mg/kg - with IIIrd grade (dose calculation as for prednisolone).
- Treatment of hyperthermic syndrome: paracetamol, ibuprofen, lytic mixtures (1ml of 2.5% aminazine + 1 ml of 2.5% diprazine are diluted to 10ml with 0.5% solution of novocaine and 5% glucose solution. Single dose of lytic mixture is 0, 1-0,15ml/kg (the mixture can be administered up to 4 times a day) For carrying out neurovegetative blockade 2,5% solution of aminazine was used.
- Anticonvulsant therapy (diazepam, sodium oxybutyrate, barbiturates, phenytoin).
- With the growth of intracranial hypertension, brain edema we shoul provide the following:
- location of the bed with raised head end 300;
- lung ventilation;
- control of osmolarity of blood plasma (no more than 300-310 mosmol / l);
- normoglycemia;
- effective cardiac output or a slight increase in blood pressure;

• if the hemodynamics is stable, administration of mannitol and furosemide.

Treatment of meningitis

• Strict bed regimen to a stable normalization of body temperature, the disappearance of meningeal syndrome and the normalization of blood, cerebrospinal fluid, for an average of 10-14-21 days. Diet therapy: infants of the first year of life are prescribed breast milk or adapted formula for the first day 1/2-1/3 with a subsequent increase to full volume for 2-3 days. Older children are given a milk-vegetable diet (table # 5 by Pevsner) minced 5-6 times a day, with the subsequent transition to table # 2 or number 15 (depending on age) during the healing period. Drinking regimen meets the age-related daily need for fluid, taking into account the daily volume of intravenously administered solutions.

• Antibacterial therapy. For meningitis of low severity or associated with meningococcal infection, the starting antibiotic may be cefotaxime 100mg/kg/day (penicillin 300-500t U/kg/ day). If a patient with Meningitis has signs of ITS, the starting antibiotic should be chloramphenicol 100mg/kg/ day (before withdrawal of the patient with TTS). In severe forms of meningitis at the first stage of therapy (until the detection of the pathogen) the drug of choice is ceftriaxone 100mg/kg/day, or cefotaxime 200mg/kg/ day. In infants younger 1 month. Of life: ampicillin 150-200mg/kg/ day (modern - flemoxin) in combination with 3rd generation cephalosporins or aminoglycosides (amikacin 15-30mg/kg/day, netilmicin 6-9mg/kg/ day). In severe cases, it is combined with fluoroquinolones (levofloxacin, flacin, ciprofloxacin, gatifloxacin) or carbapenems (named, meron). In 24 to 48 hours after the beginning of therapy, a control lumbal puncture is performed to control the effectiveness of the initiated therapy. The criterion of effectiveness is the reduction of pleocytosis by at least 1/3. If the etiological cause of the disease is detected, the starting antibiotics can be replaced by others, according to the sensitivity of the pathogen. However, in the presence of pronounced positive dynamics, namely the reduction of intoxication syndrome, normalization of body temperature, the disappearance of

meningeal symptoms, significant reduction of pleocytosis, improvement of general blood test), it is advisable to continue the initial therapy. In the absence of positive dynamics from the starting therapy for 48-72 hours, the reserve drugs are meropenem 120mg/kg/day, cefepim 100mg/kg/ day, vancomycin 60mg/kg/day. Duration of antibacterial therapy should be on average: for meningococcal and Influenza-meningitis - 7-10 days; for pneumococcal - 10-14 days; for streptococcal and listeriosis - 14-21 days; for meningitis caused by gram-negative bacilli - 21 days; for staphylococcal, enterococcal - 28 days. The criterion for the cancellation of antibiotic therapy is sanitation of the cerebrospinal fluid. Control lumbal puncture is performed after steady temperature normalization, disappearance of clinical signs of meningeal syndrome, normalization of the general blood test. Antimicrobial therapy is discontinued if the number of cells in 1 μl of liquor does not exceed 50 due to lymphocytes. In case of recurrent purulent meningitis it should be appointed a second course of reserve antibiotics (meropenem, ceftazidime, vancomycin, sulperazone). If the etiology of meningitis is established, then an anti-meningococcal gamma globulin or an anti-meningococcal plasma is administered (injected intramuscular, endolumbal). Staphylococcal etiology - antistaphylococcal plasma, gamma globulin (obtained by maternal immunization). Also use sulfonamides of prolonged action - sulfomonomethoxy - 40-50mg/kg orally once a day.

3. Detoxification therapy is carried out with 5% glucose solution in combination with 7.5% potassium chloride solution, saline solutions (isotonic sodium chloride solution, Ringer's solution), hydroxyethyl starch (reforman, stabilizol, volekam). The total daily volume is no more than 2/3 of the physiological need (with normal diuresis and with no signs of initial dehydration). Out of it the infusion volume should not exceed 1/2 of the physiological need (80ml/kg). From the second day we support the liquid deficiency in the mode of zero water balance. The volume of infusion is 1/3 - 1/2 of the physiological need. In the

case of oliguria or anuria, the introduction of fluid is contraindicated until the restoration of the diuresis.

4. Furosemide, mannitol is used for the purpose of dehydration.

5. Dexamethasone is prescribed to prevent neurosensory hearing loss at a daily dose of 0.15mg/kg every 4 hours for the first 2 days. The first dose of dexamethasone should be given 10-30 minutes before the administration of the antibiotic.

6. In viral meningitis, angioprotective agents (actovegin, trental, instenone, ceroxone) have been indicated along with infusion therapy to improve microcirculation. Apply ribonuclease - course 2 weeks 6 times a day, children under 1 year - 3mg, 2-3 years - 5-9mg, 6-10 years - 14mg, 11-15r.-20mg.

7. For herpes virus meningitis - acyclovir, for cytomegalovirus - ganciclovir, groprinosin, cimive in age doses.

8. For tubemeningitis - use different combinations of anti-tuberculosis agents: isoniazid, rifampicin and pyrazinamide for 2-3 months. The next 7 months use isoniazid and rifampicin. When the effect is not sufficient, streptomycin should be added. Duration of treatment 18-24 months.

9. Anticonvulsant therapy (phenobarbital, diphenin, sibazone, BBB. If in the anamnesis there were convulsions nootropic drugs are not used.

Here is an example of a clinical case: boy A., 1year 1month. (medical card No. 1117), who was hospitalized in the in-patient department in the POKIL in July 2012. with a diagnosis of Acute serous meningoencephalitis, basal-trunk form, severe course (isolated tuberculosis mycobacteria). Convulsive syndrome. Congenital heart defect (opened oval window) - "D" residence records. Reactive liver changes.

Complaints on admission: vomiting 4 times, flaccidity, loss of consciousness. **History of the disease:** from the words of his mother we have found out that the boy became ill acutely at night when vomiting appeared, weakness began to arise. Two weeks ago, the child had a subfebrile fever for several days. He was hospitalized at a regional hospital. The next day the

condition worsened, there was a single vomiting, loss of consciousness, strabismus appeared, forced meningeal position of the body. With the aid of medical aviation, he was taken to the ICU resuscitation unit. **History of life:** a child from 1st pregnancy, 1st labor, on time, weight at birth – 30-50g. In the maternity hospital he was diagnosed with blood tumor of head. Growing up and developing according to age. Past diseases: ARVI. Breastfeeding was up to 6 months. Objective status: severe, falccid, forced posture. Palpebral fissures D <S, pupils D = S. Meningosigns - rigidity of occipital muscles, a positive Kernig symptom, high muscle tone. Oriental strabismus was noted. Permanent convulsions were observed according to the type of myoclonia of the right lower extremity. Tendon periosteal reflexes D <S. The skin is pale, pronounced vascular reticulum on the temples. Fauces is pink, tongue is not coated. There were foaming saliva from his mouth. Puerile breathing was auscultated, the rhythm of the heart was correct, the tones muted were a little. BP - 32 per minute, HR - 120 beats per minute, blood pressure - 95-60mm/hg. Stomach is of normal configuration, palpable. The liver protruded from the edge of the costal arch at 2 cm, the margin is soft. It was hard to palpate the spleen. Physiological dispatches are issued 2 times, urination is not disturbed.

Examination: GBT - er-3,8 * 10^{12}/l, leu-44,5 * 10^{9}/l, HB-115g/l, ESR-8mm/h, trombocytes-220*10^{3} / l, eos-1%, lymph-40%, mon-6%, glucose – 4.9mmol/l. In the biochemical study of blood - bilirubin 10.0μmol/l, total protein - 64g/l, residual nitrogen 20mmol/l, urea - 3.4mmol/l, creatinine - 69μmol/l, ALT - 0.68mmol/h/l, AST - 0,45mmol/h/l, prothrombin index 80,6%; plasma fibrin - 3.55g/l. ZAS without pathology. Helminths eggs were not found. ECG - sinus rhythm, vertical position of the electrical axis of the heart. Disturbances of processes of repolarization on the back wall of the left ventricle. Ultrasound of the abdominal cavity - no structural changes were detected. In the study of cerebrospinal fluid - protein-5,87g/l, cytosis - 181 in 1 μl, lymphocytes predominate, glucose - 0,6mmol/l, erythrocytes - 20-30 in the field of view, after exposure for 15 minutes we noticed a film. In a computer examination of the

brain - internal hydrocephalus, cerebral edema, dyscirculatory encephalopathy. In bacteriological examination of saliva - isolated tuberculosis mycobacterium. In the study of feces on the intestinal group - pathogenic flora was not isolated.

The child was examined by the specialists: an ophthalmologist who diagnosed eastern strabismus, the neurosurgeon found the absence of neurosurgical pathology, the tuberculosis coordinator confirmed acute tuberculous meningoencephalitis, the neurologist found out the internal hydrocephalus.

The following **treatment** was prescribed: performed the dehydration and detoxification therapy (25% magnesium sulfate, lasix, polyionic solutions, dexamethasone) and antibacterial and antifungal therapy (ceftriaxone, fluconazole). To improve the microcirculation of the brain, pentoxifylline was used in 0.9% saline. For the prevention of DIC, heparin. Specific therapy is isoniazid, rifampicin, ethambutol. Symptomatic therapy was also conducted.

After the therapy in the ICU POKIL, the patient's condition improved, there were we noted liquor sanitation, disappearance of meningeal syndrome, hypertension, normalization of temperature, but further treatment on the basis of the regional TB dispensary was recommended.

Control questions:

- Give a definition of meningococcemia.
- What types of meningitis do you know?
- What measures are recommended at the pre-hospital stage in case of suspected meningitis?
- What is the drug of choice for the antibacterial therapy in meningitis?
- How to calculate infusion volume for purulent meningitis?

INTENSIVE CARE IN ACUTE INTESTINAL INFECTION

All acute intestinal infection (AII), regardless of etiology, have many similar manifestations. It is manifested either as a general infectious (general toxic) syndrome or as local symptoms associated with the lesions of different parts of the digestive tract (gastritis, enteritis, colitis, gastroenteritis, gastroenterocolitis, enterocolitis). General toxic syndrome in AII in children is manifested in the form of intoxication or toxicosis (more often toxicosis with exicosis), both of which have the nature of a non-specific reaction of the body to an infectious agent. The term intoxication should be understood as a primary violation of intracellular metabolic processes in combination with a lack of functional status of physiological systems aimed at the elimination of toxic products of exchange by the liver, kidney, lungs, reticulo-endothelial system.

Clinical manifestations of intoxication are lethargy, weakness, malaise, loss of appetite, which sometimes leads to the anorexia, temperature response, impaired function of various organs and systems. During the intoxication, there is the predominance of the metabolic disorders and symptoms of irritation of the parasympathetic nervous system. In toxicosis in combination with excitosis, the predominant disorders are the metabolic disorders associated with dehydration and electrolyte loss. Depending on the amount of water lost by the body, there are three degrees of toxicosis with excitosis:

- mild degree of dehydration - loss of fluid up to 5% of body weight;
- moderate degree of dehydration - loss of fluid from 5% to 10% of body weight;
- severe dehydration - loss of fluid over 10% of body weight.

The degree of dehydration of the child is determined on the basis of two parameters: the weighing of the patient with the determination of weight deficiency (or relatively to the mass that was before the disease, or relatively to that which should be in the child of this age) and on the basis of clinical manifestations of dehydration.

Table 4

Clinical assessment of dehydration

Indicators	1st degree	2nd degree	3rd degree
Weight loss	0-5%	5-10%	10-15%
Diuresis	slightly reduced	reduced	sharply reduced
Thirst	moderate	sharply expressed	absent
Skin	no changes	flaccid	wrinkled
Turgor	saved	reduced	sharply reduced
The mucous membranes	wet	dry	dry, hyperemic
Vertex	physiological	slightly depressed	depressed
HR	age standard	moderate tachycardia	embryocardia
Heart tones	loud	weakened	significantly weakened
Circulation	no changes	mild acrocyanosis	Marbling
CNS condition	No changes	flaccidity, excitation	sharp flaccidity, loss of consciousness

According to the content of electrolytes in the blood of patients with excitosis, in particular sodium, which is part of the extracellular fluid of the body and its osmolarity, there are three types of dehydration:

- hypertonic, hyperosmolar, hypernatremic, water-deficient type;
- hypotonic, hypoosmolar, hyposodium, salt-deficient type;
- isotonic type, no electrolyte disturbances.

Metabolic disorders are monitored according to changes in CBS, electrolytes, and gas composition of venous blood (Table 5).

Clinically, the dehydration according to each type of excitosis is manifested by the following symptoms, which are presented in Tables 4,6-8.

The hypertonic (water-deficient, cellular) type develops if water loss prevails, which is promoted by vomiting and rare watery stools against the background of hyperthermia and dyspnoe. Water loss is manifested by the increase in the concentration of electrolytes in the extracellular fluid (blood plasma and interstitial fluid), mainly due to hypersodiumemia, which leads to the transition of the fluid into extracellular space to equalize osmotic pressure and cause the intracellular dehydration.

Table 5

Indicators of ABB and respiratory process in children with intestinal toxicosis

Indicator	Toxicosis of 1st degree	Toxicosis of 2nd degree	Toxicosis of 3rd degree	Healthy children
pHBE, mmol / l	7,37±0,003	7,25±0,009	7,14±0,025	7,39±0,006
pCO2, mmol / l	5,3±0,36	8,9±0,38	14,3±0,75	1,2±0,12
p of venous blood	39,1±0,18	32,3±0,86	28,1±0,63	2,5±0,79

The hypotonic (saline-deficient, extracellular) type of dehydration develops gradually, a little later, in severe forms of AII with the predominance in clinical symptoms of multiple vomiting on the background of diarrheal syndrome. With increasing levels of excitosis and loss of electrolytes (mainly potassium), vomiting is unrelated to the consumption of foods or drinks, contains impurities of bile, sometimes blood (in the form of "coffee grounds"). The loss of salts is accompanied by a decrease in plasma osmolality after the transition of water and sodium from the vascular flow into the cells - intracellular hyperhydration develops, a significant increase in the amount of sodium in the cell and a decrease in the amount of potassium, hematocrit is increased. Plasma volume decreases, blood clotting occurs, blood flow is slowed down, hypoxemia, hypoxia, acidosis, microcirculation is disturbed.

In the case of presence of potassium deficiency in the blood serum the flaccidity develops, paresis of the intestine, hypotension in the child. The amount of potassium according to the standard is 4.5mmol/l. Metabolic acidosis is manifested by the clinical symptom complex: "marbling" of the skin, hyperthermia, dyspnoe, adynamia, oliguria, disorders of the peripheral circulation.

Table 6

Clinical and laboratory evaluation of hypertensive type of dehydration

Indicators	1st degree	2nd degree
Consciousness	not disturbed or excited	Sleep drunkenness, sopor
Temperature	subfebrile, febrile	febrile
Seizures	no	clonic-tonic
Thirst	moderate	sharply expressed
Skin	dry, warm	pale, dry, warm, wrinkled
The mucous membranes	dry	dry, bright
Tongue	wet	dry, red
Voice	no changes	hoarseness
Vertex	not depressed	depressed
Turgor	no changes	reduced
Eyeballs	no changes	soft
Breath	no changes	tachypnoe
HR	moderate tachycardia	significant tachycardia
BP	standard	increased
CVP	standard	standard
Paresis of the gut	absent	absent
Diuresis	reduced	oliguria
Plasma sodium	148,6±0,24	159,6±2,6

Table 7

Clinical and laboratory evaluation of hypotonic type of dehydration

Indicators	1st degree	2nd degree	3rd degree
Consciousness	Sleep	Sleep drunkenness coma of the 1st degree	Coma of the 2nd -3rd degree
Temperature	subfebrile	subfebrile, hypothermia	hypothermia
Seizures	absent	clonic-tonic	clonic-tonic
Thirst	absent	absent	absent
Skin	pale, cold	acrocyanosis, cold	widespread cyanosis, cold
The mucous membranes	wet	dry, bright	dry, bright
Tongue	coated	dry	dry, red
voice	no changes	impaired	aphony
vertex	slight reduction of the bone margin	depressed	indrawn
Turgor	no changes	reduced	sharply reduced
eyeballs	soft	Depressed	depressed
Breath	no changes	Tachypnoe	paradoxical breathing
HR	significant tachycardia	moderate tachycardia, bradycardia	bradyarrhythmia
BP	reduced	low	Less than 35-40 hg.
Diuresis	Oliguria	oligoanuria	anuria
Plasma	131,5±0,14	127,4±0,14	117,5±0,89

Table 8

Clinical and laboratory evaluation of isotonic type of dehydration

Indicators	1st degree	2nd degree	3rd degree
Consciousness	Not impaired	Sleep drunkenness, coma of the 1st degree	Coma of the 2nd -3rd degree
Temperature	subfebrile	subfebrile, hypothermia	hypothermia
Seizures	Absent	Clonic-tonic	Clonic-tonic
Thirst	Moderate	Absent	Absent
Skin	Pale, cold	Acrocianosis	widespread cyanosis, cold
The mucous membranes	moderately dry	Dry, bright	Dry, bright
Tongue	Dry, viscous saliva	Dry	Dry, red
Voice	No changes	impaired	Aphony
vertex	slight reduction of the bone margin	moderately depressed	Depressed
Turgor 1	reduced 2	reduced 3	sharply reduced 4
Eyeballs	Depressed	Depresse	Depressed
Breath	Not impaired	Tachypnoe	paradoxical breathing
HR	significant tachycardia	moderate tachycardia, bradycardia	bradyarrhythmia
BP	Standart	Reduced	low
Diuresis	Reduced	Oliguria	oligoanuria
Plasma sodium	standart	standart	standart

Isotonic (general) type of dehydration develops due to the extracellular and intracellular proportional loss of water and electrolytes. Most commonly found at the beginning of AII. Because water and electrolytes are lost in physiological proportions, this condition is compensated more quickly in the course of treatment than the two previously mentioned types of excitosis.

The results of the study of the level of electrolytes have differential diagnostic value. The degree of excitosis does not always correspond to the severity of the condition of the child. Sometimes clinical symptomatology of the 2 degree of dehydration of the isotonic type of excitosis causes hypovolemic disturbances in the body of the patient.

Rehydration therapy

The basis of treatment of AII in children is timely and adequate rehydration therapy - compensation for water and electrolyte losses. Proper rehydration therapy is a priority in the treatment of AII, both for secretory and invasive diarrhea. Nowadays, rehydration is divided into oral and parenteral. To perform the rehydration, it is necessary to define:

- The daily need for fluid.
- Type and degree of dehydration.
- Current pathological losses.
- The total level of fluid deficiency.
- Determine the method of rehydration.

Procedure

I. Determine the degree of excitosis. To do this, you need to know the lack of fluid, which is calculated on the percentage of reduction in body weight from the moment of illness onset to the time of inspection (1st stage - body weight reduction up to 5%, second stage. - up to 10%, 3rd stage - up to 15%). If the body weight before the disease is unknown, the degree and type of dehydration is determined by clinical features (Table 2). It is even easier - in this case, to set the fluid deficiency by 10%.

• Determine the method of rehydration therapy. In the excitosis of 1st-2nd stages. In the case of absence of uncontrolled vomiting and severe anorexia, oral rehydration may be sufficient.

Oral Rehydration (OR)

Oral rehydration in acute intestinal infection should be the first treatment that is performed at home in the condition of the onset of the first symptoms of the disease. OR made in time makes it possible to effectively treat most children at home and to reduce the incidence of severe excitosis.

Procedure

- Apply solutions: "Glucosolan", "Oralite", "Gastrolite" - 1st generation; Rehydron - IInd generation; ORS 200 HIPP - 3rd generation. You should not use fruit juices, sugary sodas.
- 2. Rehydration is carried out in 2 stages:

• Stage I - recovery of the volume of the lost fluid: in case of excitosis of the 1st stage. - volume of liquid - 50ml/kg of body weight, in case of esicosis of the second stage. - 100ml/kg of body weight. Stage I lasts for 4-6 hours. Estimated for 1 hour of administration, the volume of the solution is poured into a graduated utensil, given to the baby by a pacifier, using a pipette, a teaspoon every 5-10 minutes. In case of refusal to drink or vomiting, the solution is injected through the nasogastric tube dropwise with a 10 ml / min intravenously. Length from the ear to the nose + from the nose to the xiphoid appendix.

Efficiency evaluation, further tactics

a. Rehydration is effective (disappearance of thirst, improvement of turgor of tissues, elasticity of skin, moistening of mucous membranes, increase of diuresis, disappearance of signs of disturbance of microcirculation) - transition to the second stage (maintenance therapy).

b. Rehydration is not efficient enough (signs of dehydration persist) - continue similar treatment for another 4-6 hours.

c. Rehydration is ineffective (increasing dehydration, persistent vomiting, profuse diarrhea, increasing symptoms of toxicosis) - switch to the infusion

therapy (parenteral rehydration) by puncture of the peripheral vein or catheterization of the main vein.

Stage II of the OR - supportive therapy. If the number of stools is no more than one every 2 hours, or at least 5ml of liquid feces per 1kg of body weight per 1 hour, the volume of fluid is 5ml/kg of body weight / hour. In severe diarrhea, fluid volume = 10ml/kg of body weight / hour. The calculated volume is distributed evenly throughout the day.

In order to avoid complications (tissue sponginess, decrease in diuresis), especially in children with concomitant pathology of neurotoxicosis, marked by colitis, it is advisable to substitute up to half of the liquid with strong sweetened (3% sugar) tea, preferably green, with lemon, rosehip, fruit (apple) decoction. For 24 hours of the oral rehydration period, the patient's body weight should be increased from 5% to 10%.

Contraindications for the oral rehydration

- Severe dehydration (more than 10% weight loss in young children and more than 6% older).
- Vomiting that lasts for 2 hours of oral rehydration.
- Intestinal paresis.
- Stupor, coma, infectious-toxic shock.
- Oliguria and anuria that do not disappear during the first rehydration phase.
- Metabolic alkalosis.
- Inefficiency of oral rehydration during the day.

In the case of excitosis of the 3rd stage, uncontrollable vomiting, anorexia, refusal to drink, rehydration therapy is started with parenteral (intravenous) injection of fluid (infusion therapy), combining it with oral rehydration.

Parenteral rehydration (infusion therapy).

Procedure

- Assess the degree of excicosis. If the body weight to the disease is not known, then its loss as a result of dehydration is taken for 10%.

• Determine the type of excicosis. Prior to laboratory confirmation you should take into account the clinical manifestations (Table 4,5,6). As a last measure, you can evaluate the situation as an isotonic type of excicosis.

• Calculate the daily volume of the necessary fluid for the child (according to J. Dennis method): I degree of excicosis: under 1 year - 140-170ml/kg; 1-5 years - 100-125 ml/kg; older children - 75-100 ml/kg.

II degree of excicosis: under 1 year - 160-180ml/kg; 1-5 years - 130-170 ml/kg; older children - 110 ml/kg.

III degree of excicosis: up to 1 year - 200-220 ml/kg; 1-5 years - 170-180 ml/kg; older children - 120-150 ml/kg.

Calculate the daily amount of infusion:

I degree of exicosis - up to 40% of the total daily volume of fluid; II degree of excicosis - up to 60% of the total daily volume of fluid; III degree of excicosis - up to 80% of the total daily volume of fluid.

If the child has pneumonia, the infusion volume should not exceed 50% of the required daily volume.

Calculate the volume and duration of the first infusion (fraction). The calculated volume of infusion should be administered within the day. However, in the case of limited accessibility to the main (subclavian, etc.) vein, fluid should be injected into the peripheral veins according to the fractional method.

I degree of excicosis: lasting at least 4 hours, you can administer full dosage of calculated volume (per day).

II degree of excicosis: duration of the infusion (I fraction) not less than 6 hours, administration of 1/2 of the full dosage of calculated daily volume of infusion; in 8-12 hours perform the second infusion (fraction).

III degree of excicosis: duration of infusion (I fraction) not less than 8 hours, administer 1/2-2/3 of the full dosage of calculated daily volume of infusion; in 8-12 hours perform the second infusion.

Calculate the rate of fluid injection.

Volume of fluid in ml / min. = total volume of fluid (ml): (3 × hours (min) of infusion). For infants, the rate of intravenous injection is not more than 60-80 ml/h (not more than 14-16 drops / min).

Select solutions, determine its ratio and the sequence of its introduction.

• Optimal crystalloid solutions for parenteral rehydration in infants are 5% glucose solution and 0.9% sodium chloride solution. Colloidal solutions: plasma, 5-10% albumin solution, reopolyglucine, stabilizol, reforman.

• Generally, intravenous infusion is started with colloidal solutions equal to 1/3 of the total infusion volume. The daily volume of colloidal solutions is distributed by the number of infusions. The dose of the drug at 1 injection should not exceed 15ml/kg body weight. Rehydration of children with I-II degree of exicosis, followed by repeated vomiting, is advisable to carry out by glucose-saline solutions without the use of colloids.

• In the same way, a daily amount of 5% glucose solution and 0.9% sodium chloride solution is distributed between the fractions.

• In the case of isotonic excicosis, the ratio of volumes of 5% glucose solution to 0.9 NaCl solution equals 2: 1 (these solutions can be mixed in one vial and can be administered simultaneously).

• In the case of hypertonic type of exicosis, the ratio of volumes of 5% glucose solution to 0.9% NaCl solution equals 3: 1 (if these solutions are not mixed in one vial, then the infusing is stared with glucose solution).

• In the case of hypotonic type of exicosis, , the ratio of volumes of 5% glucose solution to 0.9% NaCl solution equals 1: 1 (if these solutions are not mixed in one vial, then the infusing is stared with NaCl solution).

• Potassium is an essential component of infusion therapy. In hypertonic type of exicosis potassium is added to the infusion solutions in the form of 7.5 solution in a dosage of 1-2 ml/kg per day; in the case of isotonic and hypotonic - 3ml/kg a day. In these situations, it is possible to administer panangin (asparks) in

a dosage of 1ml/kg per day. The daily dose of potassium preparations is evenly distributed between the fractions throughout the day. The concentration of potassium chloride in the infusion is less than 0.5%. Contraindications for intravenous administration of potassium - anuria or pronounced oliguria (less than 20 ml of urine per kg of body weight per hour).

• Add 1-2 ml/kg/day of 10% solution of calcium gluconate and in the case of III degree of exicosis, pronounced disturbances of microcirculation, toxic, acidotic breathing, disorders of consciousness - choose 4% solution of sodium bicarbonate in a dosage of 4ml/kg/day. The calculated amount of sodium bicarbonate is distributed for 3-4 injections and injected intravenously dropwise with glucose solution.

Evaluate the effectiveness of therapy. Proper infusion therapy is followed by:

• Elimination (reduction) of signs of excicosis within the first 24 hours (criterion - weight gain by 7-9% for the first day).

• Stable improvement of hemodynamics (criterion - normalization of blood pressure, heart rate).

• Restoration of microcirculation (color improvement, normalization of the skin temperature, stabilization of hour diuresis).

• Improving the overall condition of the child.

• Calculate the amount of rehydration therapy for the following days: Take into account residual deficiency of the body weight.

• Take into account the daily need for the fluid and its current pathological losses: with hyperthermia above 38° C, the fluid is added at the rate of 10ml/kg/day; with dyspnoe for every 10 breaths above standard 10ml/kg/day; at vomiting and diarrhea - 20-30ml/kg/day; in the case of the reduction of diuresis - 30ml/kg/day. Age daily requirement for water: under 6 months - 120-100ml/kg; 6 months - 2 years - 100-80ml/kg, older than 2 years - 80-40 ml/kg.

In all cases, you should be aimed to move from parenteral rehydration to oral maintenance rehydration therapy as quickly as possible.

Detoxification therapy

It is carried out in the presence of severe intoxication syndrome and infectious toxicosis. In mild forms, the use of oral rehydration solutions is sufficient. In moderate and severe forms, intravenous administration of 5% glucose solution, 0.9% sodium chloride solution, colloidal solutions (albumin, reopoliglyukin, reforman, stabilizol) simultaneously with oral rehydration. The total infusion volume is 50-100ml/kg/day. The ratio of colloids to crystalloids is 1: 2. It is advisable to start infusion with the introduction of colloidal solutions in a single dose up to 20ml/kg. Then glucose and sodium chloride solutions are introduced in a 1:1 ratio. The total volume of fluid, including rehydration, should not exceed 150-160 ml/day.

Enterosorption

- **Enterodesis.** The solution is made up immediately before use (1 teaspoon of the powder is dissolved in 100 ml of cooled boiled water). For children per os: under 1 year - 20 ml/day, 1-3 years - 30ml/day, older than 3 years - 50/ ml/day. The daily dose is distributed for 3-4 doses.
- **Polyfepan.** Available as a powder, always in moist form. Infants are prescribed 1 teaspoon, the elder children - 1 dessert spoon 2-3 times a day.
- **Smecta.** Available as powder in sachets. It is diluted with water before use. Children under 1 year are prescribed 1 sachet a day, older ones 2-3 sachets.
- **Enterosgel.** Available as powder. Infants are prescribed 1 teaspoon, older children - 1 dessert spoon 2-3 times a day, pre-dissolved in 50-100ml of cooled boiled water.

These listed above drugs are most often used in domestic pediatric practice, the first place is given to Smecta. The duration of enterosorption in **AII** is 5-7 days. The criterion for early cancellation is normalization of stools or its absence within 2 days.

Control questions:

- What diarrhea is classified as secretory?
- What is invasive diarrhea?
- How to calculate the daily amount of rehydration with AII?
- What is the physiological need of a baby in fluid at 11 months of age?
- What should be prescribed for salt-dehydration in baby with AII?

CYCLIC VOMITING SYNDROME (CVS). INTENSIVE CARE.

The cyclic vomiting syndrome is a set of symptoms caused by the increased content of ketone bodies in the blood: acetone, acetoacetic acid and β-oxybutyric acids.

Pathological changes in the body leading to the development of CVS:

- metabolic disorders;
- reducing glucose uptake;
- activation of gluconeogenesis;
- increased lipolysis;
- increased level of free fatty acids;
- increased level of fatty acids decomposition products in the blood (acetone, acetoacetic acid and β-oxybutyric acids).

Ketonemia in the body is accompanied by: metabolic acidosis, vasoconstriction, hypovolemia, hypocapnia and hypoglycemia, which leads to irritation of the gastrointestinal mucosa in the form of spastic pain and to toxic effects on the nervous system.

There are primary and secondary cyclic vomiting syndrome. Primary acetonemic syndrome occurs in children with neuro-arthritic diathesis between of 2 and 10 years of age. Secondary CVS is caused by the following diseases: diabetes, Itsenko-Cushing's disease, hyperinsulinism, thyrotoxicosis, infectious toxicosis, cerebral trauma, brain tumors.

Clinics of cyclic vomiting syndrome: repeated vomiting (1-5 days), dehydration and metabolic intoxication, pale skin, red cheeks, excitation is followed by flaccidity, drowsiness, phenomena of meningism, disorders of hemodynamics, spastic pain in the abdomen, nausea, liver enlargement up to 1-2 cm, subfebrile temperature, smell of acetone from the mouth. In the biochemical analysis of blood, there is an increase in ketone bodies, hypochloremia, acidosis, hypoglycemia, hypercholesterolemia, an increase in β-lipoproteins; in the general blood test - leukocytosis, a slight increase in ESR.

Acetonemic crisis precursors:

- anorexia;
- lethargy;
- excitation;
- migraine headache;
- nausea, pain in the umbilical region;
- acholic feces;
- the smell of acetone from the mouth.

Intensive care of CVS

- Stomach and intestines lavage and with 2% soda solution;
- oral rehydration;
- antispasmodics - drotaverine 10-20mg 3 times a day (1-6 years), 20-40mg (schoolchildren);
- Enterosorbents;
- infusion therapy - 0.9% saline, 5% glucose solution (1:1), reosorbylactate 10ml/kg, xylate (acetone in urine 4+) 20ml/kg, glucosyl 20-60ml/kg. The total volume of infusions is 50ml/kg/day. Add 5% solution of vitamin C 3 ml / day, cocarboxylase 5-100mg/day, with hypokalemia 5% potassium chloride 1-3 ml in 100ml of glucose. In case of vomiting 1-2 times metoclopramide not more than twice a day 0,1mg/kg for children under 6 years; 6-14 years - 0.5-1.0ml;
- in the further treatment use enzymes, hepatoprotectors 1-1,5months, advise the patient to keep to the diet.

Control questions:

1. What is cyclic vomiting syndrome?
2. What are the pathological changes in the body with cyclic vomiting syndrome?
3. What are the urgent measures for child of 2 years with cyclic vomiting syndrome (acetone in urine 4+)?

LIVER FAILURE IN VIRAL HEPATITIS

The major cause of liver failure is hepatodystrophy on the background of viral hepatitis (B, C, D). Therefore, the authors believe that to facilitate the understanding of the presented material, it is necessary to mention the epidemiology and basic diagnostic criteria for hepatitis.

Table 9

Epidemiological signs of viral hepatitis

Virus		**Sourse**	**Ways of spreading**	**Stages**			
				incubatory	prodromal	icteric	reparative
HAV	RNA Enteroviruses	Contaminated, carrier	Fecal-oral, domestic	10-45 days	3-5 days	2-4 weeks	2-3 months
HBV	DNA hepadnoviruses	Contaminated, healthy carrier	Parenteral	6-26 weeks	5-7 days	3-4 weeks	6-10 months
HCV	RNA flaviviruses	Contaminated, healthy carrier	Parenteral	6-8 weeks	7-8 days	3-6 weeks	6-12 months
HDV	Defective RNA	Contaminated, carrier	Parenteral	2-24 weeks	5-7 days	2-8 weeks	6-12 months
HEV	RNA calicy viruses	Contaminated carrier	Water	18-130 days, 3-28 weeks	1-12 days	2-3 weeks	6-12 months
HGA	RNA flaviviruses	Contaminated carrier	Parenteral	7-11 days	5-7 days	3 weeks	6-12 months
HTV	DNA cisroviruses	Contaminated carrier	Parenteral				6-12 months
HFV	RNA	Contaminated carrier	Parenteral				6-12 months
HSV	Cisroviruses	Contaminated carrier	Parenteral				6-12 months

Table 10

Clinical differential diagnostic features of viral hepatitis

Sign	A	B	C	D	E
Incubatory	14-25 days	2-6 months	2-12 months	2-24 months	15-45 days
Onset	acute	gradual	gradual	acute	acute
Intoxication during pre-icteric period	pronounced	slightly pronounced	slightly pronounced	pronounced	pronounced
Intoxication during icteric period	slightly pronounced	pronounced	absent or slightly pronounced	pronounced	absent or slightly pronounced
Allergic rash	absent	sometimes	sometimes	sometimes	absent
Sererity	light or mild	mild	light or non-icteric	severe and malignant	light
Jaundice	1-1.5 weeks	3-5 weeks	about 1 week	2-8 weeks	1-2 weeks
Chronization	absent	frequently	up to 50%	frequently	absent
Serological indicators	anti-HAV IgM	HBsAg, HBcAg, anti-HBc IgM	anti-HCV, RNA HCV	HBsAg, anti-Bc, anti-HDV IgM	anti-HBV

Hepatic insufficiency

There are acute and chronic liver failure, which are divided into three stages: the initial (compensated), the onset of clinical manifestations (decompensated), the terminal (dystrophic). The terminal stage of liver failure leads to hte hepatic coma. Hepatic coma – is a state of sharp inhibition of higher nervous activity, manifested by deep loss of consciousness, impaired mobility, sensitivity, reflexes, absence of response of the child to sound, light and other stimuli and accompanied by widespread hepatocyte necrosis, which leads to impaired function of the liver and general body toxicosis.

The clinical picture. In the initial stage of liver failure, weakness, increased fatigue, decreased appetite, dyspeptic symptoms, sleep disorders, headaches, digestive disorders, nausea, vomiting, anorexia, fever, development of anemia, thrombocytopenia are observed. In the peak of liver failure, there is a decrease in liver size, "liver" smell from the mouth, signs of portal hypertension - ascites and edema. In the case when the hepatocytes are damaged it impairs the production of plasma albumin by the liver, inactivation of aldosterone, leading to secondary hyperaldosteronism, increased reabsorption of sodium ions into the extracellular space. In addition, of great importance is the production of the anti-diuretic hormone that causes the development of edema.

In chronic liver failure, endocrine changes gradually occur. In boys: testicular atrophy, gynecomastia, hair loss under the armpits, on the head, as for girls: atrophy of the uterus, mammary glands, menstrual cycle is disrupted. All these changes are caused by the accumulation of estrogens due to insufficient inactivation of the liver. Disruption of inactivation of estrogens
and some vasoactive substances to small telangiectasias - vascular "stars," palmar erythema, enlargements of the vascular reticulum on the face. Anorexia, significant weight loss and polyhypovitaminosis gradually develop Often, liver failure is accompanied by impaired renal filtration ability and the development of azotemia. In the pre-comatose state there is anorexia, nausea, reduction in liver size, jaundice, hyperbilirubinemia, amino acids and lactic acid in the blood. In the future, increase number of mental disorders, depression, and sometimes euphoria, irritability, impaired memory, sleep, tendon reflexes, meningeal signs, respiratory distress, pulse becomes arrhythmic, body temperature decreases ‘liver’ smell from the oral cavity, hemorrhagic phenomena are exacerbated. Subsequently, ESR, residual nitrogen and ammonia levels are increased, hypokalemia, hyponatremia, metabolic acidosis are noted, neuropsychiatric disorders appear: flaccidity, apathy, euphoria, insomnia, headache, memory impairment (pre-coma of 1st degree); confusion, psycho-motor excitement, changed by flaccidity, convulsive readiness, muscle

twitching, short-term seizures (pre-coma of 2nd degree); absence of consciousness, absence of reflexes, absence of painful reaction, often convulsions, dilation of pupils, involuntary urination and defecation, pathological breathing (coma). The hepatic coma often ends in death for the baby.

Urgent care for hepatic coma

- Normalization of metabolism in hepatocytes - hyperbaric oxygenation (HBO), preventive IVL;
- Increasing glycogen content in hepatocytes - 10-20% glucose solution 10 ml / kg with insulin;
- Cell membrane stabilizers - prednisolone 3-5mg kg, essentiale 3-5ml;
- Antioxidants - 5% solution of ascorbic acid, vitamin E 5mg/kg/day;
- To relieve liver cell edema - Lasix 0.2-0.4mg/kg;
- Antihypoxants - phenobarbital 1-5mg/kg, droperidol 0.1-0.2mg/kg;
- Drugs that protect membrane structures from over-oxidation - cytochrome C, lipoic acid 70mg/kg, 2% soda solution, cocarboxylase 5mg/kg/day;
- Artificial detoxification - peritoneal dialysis, enterosorption, plasmapheresis, hemodialysis, forced diuresis;
- Correction of plasma protein composition and hydro-electrolyte metabolism;
- In case of hemorrhagic syndrome hemostatic drugs are heparin 30U/kg/day under the control of coagulogram, fresh frozen plasma 10ml/kg;
- For the inhibition of ammonia production, its utilization and the rapid elimination from the body, the preparation of dufalac in a dosage of 5-20ml 3 times a day is recommended;
- In psychiatric disorders, neuroleptics are recommended - (diazepam, relanium, seduxen) 0.5% solution - 0.1ml/kg - single dose intravenously slowly.

The nature of measures in liver failure.

• *Prevention of auto-intoxication* (reduction of formation of ammonia and phenols in the intestine): washing of the stomach and intestines with 2% sodium bicarbonate solution (soda), 1% sodium bicarbonate solution is used in infants; in gastric hemorrhage use isotonic sodium chloride solution (saline); hungry-water pause for 10-24 hours, give infants drink 5-10% glucose solution for 5-10 ml every 10-15 minutes; give older children sweetened tea, 5-10% glucose solution, broth of dried briar (with 3% sugar), broth of raisins (with 3% sugar) - often in small portions; then the children of the first year of life are transferred to a dosed diet of strained breast milk, in its absence - sour milk formulas (adapted): the number of feedings per day - 10, the interval between feedings - 1½-2 hours, the amount of food per feeding 10- 20ml, night break - 6:00; in the following days, 10ml of food (100ml a day) is added to each feeding, after reaching the amount of 50 ml of food per day (500ml a day), the baby is transferred to 8 times feeding 70-80ml (560-640ml per day). and in the following days - for 7 times feeding 100-120ml (700-840 ml per day), then start 6 times feeding (if the child is older than 2 months) up to 150ml (900 ml per day) and 5 times (if the child is older than 5 months) - 180-200ml (1 liter per day); when the child reaches the repetition factor and volume of feeding, appropriate to the age standard, is introduced gradually at intervals of 2-3 days appropriate feeding in the usual sequence; during small feeding, the lack of food is filled with drinking and infusion therapy; children older than one year after a hungry pause receive predominantly carbohydrate food (less protein in food - less ammonia and phenols in the intestine): liquid milk porridge, kissels, rice broth, vegetable broth, fruit and berry juices;

Antibiotics (suppression of intestinal microflora forming toxic products - ammonia and phenols): kanamycin - 50mg/kg/day - in 4 doses, gentamicin - 3-5-7mg/kg/day - in 3 doses, tabramycin - 3 - 5-7mg/kg/day - in 3 doses; sizomycin - 3-5-7mg/kg/day - in 3 doses, polymyxin - 10mg (100 thousand units)/kg/day - in 3-4 doses.

• *For prevention of septic process*: antibiotics parenterally – intravenous and intramuscular semi-synthetic penicillins, cephalosporins of II-III generation, carbapenems, monobacts.

• *Detoxification, correction of hypovolemia, hypoglycemia, disorders of electrolyte metabolism, acid-base state, improvement of blood rheology, parenteral nutrition (infusion therapy):* the total amount of fluid injected during the day intravenously should not exceed 70% of the daily requirement; moreover, ⅔ of the total amount of infusion is a glucose solution and ⅓ is a colloidal solution; the daily volume of infusion is divided into 2-3 parts (fractions), the composition of each fraction includes glucose solution and colloidal solution; fluid is introduced drop wise at a rate of 14-16 drops in 1 min. (not more than 80 ml / h), the interval between fractions is several hours:

-glucose solution of 10%, insulin (1 unit per 4-5g of glucose); preferably in the form of a polarizing mixture, for which is added:

• potassium chloride solution 7,5% - 1ml/kg/day - the calculated amount is divided by the number of fractions, the outcome is injected into the glucose solution - the concentration in the infusion should not exceed 0,5%;

• pangangin solution - 1ml/kg/day - divided by the number of fractions, the outcome is introduced into glucose solution;

- calcium gluconate solution of 10% (1-2ml/kg/day), or calcium chloride solution of 10%;

(0.5-1ml/kg/day) - divided by the number of fractions, the outcome is injected into glucose solution;

- in case of frequent vomiting it is recommended to add 5-20ml of 10% sodium chloride solution to the composition of the polarizing mixture; after the introduction of the polarizing mixture administer <u>the colloidal solutions:</u>

-Plasma (native, fresh frozen) - 10ml/kg/single dose;

albumin solution 5-10-20% - 15-10-5ml/kg/single dose respectively;

-reopolyglukin - 10ml/kg/single dose;

- Reogluman - 10ml/kg/single dose;

-to stimulate diuresis in one of the fractions, the colloidal solution is replaced by osmodiuretics: mannitol 15-20% solution - 5-10ml/kg/single dose - intravenously dropwise, or sorbitol 20% solution - 5-7,5ml/kg/single dose - intravenous dropwise;

• in case of severe acidosis (urinary pH caused by the litmus indicator tape, is less than 6.5): sodium hydrocarbonate solution 4% - 3-6 ml/kg/single dose - intravenously dropwise (caution! threat of vascular endothelial damage, including medullary), or trisamine 0.3% solution

• 12.5 ml/kg/single dose intravenously dropwise - under the control of urine pH with a litmus indicator tape, until the raise of the pH to 6.5 - 7.0.

4. Reduction of necrobiotic, exudative and sclerotic processes in the liver: prednisolone (drug of choice) - 5-10mg/kg/day - intravenously - for 4-5 injections;

• proteolytic enzyme inhibitors: contrical, tracilol, tzalol - 1000 - 2000U/kg/day; Gordox - 10000 - 20000U/kg/day - intravenously dropwise on 5-10% glucose solution - divide the daily dose into 3 injections (every 6-8 hours).

5.*Vitaminotherapy:*

-Cocarboxylase - 5-10-15mg/kg/single dose - intravenously 2-3 times a day;

• Ascorbic acid solution 5% - 0.2-0.3ml/kg single dose - intravenously 2-3 times a day;

-Vicasol - 0, 1 ml/kg/daily dose - intramuscularly - for 2 injections.

• *Hypoxia correction:*

-inhalation of moistened oxygen (through mask, nasal catheter);

• enteral introduction of oxygen (through a gastric tube); -hyperbaric oxygenation (in the barochamber).

7. *Improvement of blood circulation and microcirculation in the liver and other tissues:*

-droperidol 0.25% solution - 0.05-0.1ml/kg/single dose - intravenously slow on 10% glucose solution, can be re-injected in 4-8 hours;

-Eufilin solution of 2.4% - 0.1 ml/kg/single dose - intravenously slowly on saline solution; -dipyridamole (quartile, persantine) - 5mg/kg/single dose - with 10% glucose solution intravenously dropwise; or trental - 5 mg/kg/ single dose - with 10% glucose solution intravenously dropwise.

8. *Termination of psychomotor excitement:* diazepam (Relanium, Seduxen) 0.5% solution - 0.1ml/kg - single dose intravenously slowly, it can be re-injected in 8 hours; or sodium oxybutyrate 20% solution - 0.25-0.5-0.75ml/kg/single dose intravenously slowly (1-2 ml / min) in 30-50 ml 5-10% glucose solution, can be re-injected in 4- 6 hours.

9. *Surgical methods of detoxification*: artificial blood transfusions, plasmophoresis, hemodialysis, hemosorption, cross-circulation with healthy donors, lymphosorption, connection of heterogeneous liver.

Control questions:

1. What is the main cause of liver failure?
2. What is the classification of hepatitis according to MK-10?
3. Write a list of the urgent measures for liver coma.

ACUTE STENOTIC LARYNGOTRACHEITIS AND DIPHTHERITIC CROUP

Acute stenotic laryngotracheitis is a syndrome of infectious disease characterized by impaired passage of inhaled air through the respiratory cleft, which is regarded as croup.

Acute stenotic laryngotracheitis (ASLT) occurs only in childhood, mainly in children under 3 years, and then its frequency decreases from 3 to 6 years and from 7 to 14 years. In children under 6 months of age, this condition does not occur. Boys fall ill three times more often than girls.

Etiology: the main cause of ASLT is: viruses - 20%, virus in combination with bacteria 45%, mycoplasma - 15%, chlamydia - 7%. adenovirus - (13.6%), respiratory syncytial - 3%. In 2005, a new virus was discovered - the boquavirus, which causes ASLT in children of 3 years of age, it is combined with bowel dysfunction (vomiting, diarrhea). The cause of acute stenotic laryngotracheitis is also infectious diseases of scarlet fever, whooping cough and others. In children from 3 to 7 years of age, ASLT may also cause recently discovered metapneumovirus, which combines croup syndrome in the clinics and inspiratory shortness of breath.

All viruses are resistant to the epithelium, but are divided into 2 groups according to ability to cause pathological process:

• with specific epitheliotropic (parainfluenza, influenza, rhinovirus, respiratory syncytial virus, bocavirus), causing pathological process with destruction of epithelial cells and causing tough morphological changes;

• viruses for which epithelial cells are the primary focus of infection at the site of the entrance gate (adenovirus, measles, rubella, herpes).

Anatomical and physiological features of respiratory organs in children:

• small size of larynx and soft cartilage, plates converge at proper angles;

• narrow, elongated epiglottis;

• voice folds are short;

• up to 6 months lymphoid tissue is formed;

• there are many glands in the mucous membrane of the upper respiratory tract;

• increased reflex excitability of the muscles that close the glottis;

• functional immaturity of the reflexogenic laryngeal zones.

Pathogenesis of the croup syndrome is

1. swelling of the mucous membrane of the larynx and trachea;

2. spasm of laryngeal and tracheal muscles;

3. hypersecretion from the glands of the respiratory mucosa.

Pathomorphologic changes are manifested by hyperemia and edema of the mucous membrane of the larynx and trachea, especially in the nasopharyngeal cavity, the accumulation of pathological content and its transformation into the crusts, especially in hyposecretory form of the disease. Microscopic examination of the mucous membrane reveals dystrophic changes of the epithelium, its desquamation, and necrotic-hemorrhagic and fibrinous-necrotic changes, if bacterial microflora is associated.

Main clinical manifestations:

• rough 'barking' cough;

• noisy, stenotic breathing;

• dysphonia and voice hoarseness.

The course of ASLT is gradual. There are compensated, subcompensated, decompensated and terminal (pre-fixation) stages. The disease onset comes suddenly, in the middle of the night, when there are shortness of breath and a dry ringing (barking) cough. There is a general excitation, children become restless, sleep poorly, refuse to eat, but at the end of the night the phenomena of laryngeal stenosis disappear, and there are attacks of dyspnoe occur again in the middle of the night and last for several days in a row. However, it happens that during the day the phenomena of laryngeal stenosis increase and sequently appear I, II, III stages of laryngeal stenosis. The appearance of signs of difficult breathing at night is explained, perhaps, by the

fact that due to the horizontal position of the baby in the infraorbital space swelling of the mucous membrane increases and there is ат accumulation of pathological content in the larynx, which contributes to laryngospasm.

At the compensated stage the child is restless, crying, poorly asleep. The breath is noisy, there is inspiratory shortness of breath, prolonged inhalation, falls or shortens of the pause between inhalation and exhalation in the case of restless behavior of the child. In a calm state there is no inspiratory shortness of breath, but an increase in cardiac activity in response to inspiratory shortness of breath occurs. In this stage, the act of breathing is restructured, providing the body with oxygen. An important role in this is the irritation of the respiratory center with carbon dioxide.

At the subcompensated stage the difficult breathing increases, the inspiratory shortness of breath is observed in the resting state, and if the child is restless, the auxiliary muscle aids breathing, manifested by the involvement of the jugular and subclavian foramens, intercostal spaces, is involved in the act of breathing. The phenomena of heart failure are growing. The chest radiograph shows an increase in the pulmonary pattern, indicating impaired circulation in the small circle.

The decompensated stage is characterized by sharply difficult noisy breathing. Not only the chest but also the abdominal muscles are involved in the inhalation process, so the epigastric region is singnificatly extended. Due to the increased work of the respiratory muscles, oxygen deficiency increases, deep acidosis develops, oxidative-redox processes are broken. Not fully oxidated metabolic products block the enzyme systems, resulting in difficult elimintaion of oxygen. Therefore, the cyanosis of the visible mucous membranes increases, the skin becomes marbled - it is a terrible sign of vascular insufficiency. Blood pressure decreases sharply, the pulse becomes weak. In the case of auscultation, respiration in the lungs is weakened, sometimes not even heard, which is caused by depression of the respiratory center.

Stage of preasphyxia *i*s characterized by shallow breathing, as Chane-Stokes, mild sites of the chest and the epigastric area are not retracted, no signs of noisy breathing. *No cough.* Heart tones are quiet, pulse is almost absent, blood pressure is not possible to determine. Cyanosis is replaced by sharp pallor, the patient becomes unconscious, the pupils dilate, enophthalmos, involuntary urination and defecation are observed. Failure to provide emergency aid timely will result in death caused by tissue respiration disruption due to hypercapnia, intoxication.

Clinical forms of croup:

• edematic- characterized by a gradual increase in severity, dry "barking" cough, not productive, there is a decrease in voice height. Children older than 2 years are in forced body position. Auscultatory - weakened breathing;

• spasmodic - the voice is disturbed moderately, the cough is "cawing." During sleep, the breath is normal, calm, and after the awakening the voice disappears. There are few or no auscultatory changes;

• hypersecretory - a cough with viscous sputum is noted, the condition worsens during sleep due to obstruction, which provokes laryngospasm.

The degree of ASLT in clinical manifestations.

• Depression of the jugular fossa, signs of respiratory failure 1, oxygen saturation (saturation) is 90%;

• perioral cyanosis, breathing rate (BR) increased to 25% above the age norm, depression of intercostal spaces, respiratory failure 2, saturation - 90% -70%. Such a child needs intensive care;

• Acrocyanosis, diaphragm retraction, BR increased to 50% above the age standart, saturation - less than 70%, respiratory failure 3. It is necessary to transfer the patient to the intensive care unit;

• asphyxia, total cyanosis, terminal status., Arrhythmic breathing, swelling of the cervical veins, BR increased to 70% above the age standard, saturation - less than 50%.

Acute stenotic laryngotracheitis should be differentiated with diphtheria of the larynx (true croup), which is characterized by a slow onset, hoarse voice, fibrinous coatings, increased difficulty in breathing; the phenomena of toxicosis, cervical lymphadenitis and tissue edema are observed. From the very beginning, a wet, not a dry, cough is noted, then when the films are formed it becomes dry. The leading symptom is aphonia in the case of diphtheria of the larynx. Finally, bacteriological examination is essential.

The course of the diphtheria of the larynx is characterized by the stages: catarrhal or dysphonic (croupous cough), stenotic (compensated, subcompensated and decompensated) and asphytic. The initial stage lasts 1-3 days, the beginning is slow, subfebrile themperature, cough is loud, hoarse voice during laryngoscopy pronounced swelling and hyperemia of the mucous membranes. The younger the baby, the faster the stenosis with aphonia and shortness of breath increases. Toxicosis, cyanosis, hypoxia increases. During laryngoscopy, on the background of the laryngeal and tendons hyperemia a gray film can be seen. This stage lasts 2-3 days. The subcompensated phase is characterized by constant stenosis, shortness of breath, noisy breathing at rest, respiratory failure. With decompensated stenosis, there is a sharp excitation, pulse wave fall on inhalation. The asphytic stage lasts for a few minutes, the breath becomes shallow, general cyanosis, single breaths, bradycardia, respiratory arrest are observed.

Acute stenotic laryngotracheitis and diphtheritic croup should be differentiated with epiglottitis (edema and inflammation of the epiglottis), pneumonia, foreign bodies in the respiratory tract, allergic stenosis, laryngospasm in children with rickets, spasmophilia. In this case, in addition to the anamnesis, the dynamics of the disease, clinical and radiological examinations, direct laryngoscopy and bronchoscopy are essential.

The prognosis for ASLT and diphtheritic croup is serious, because in some cases it leads to death, even if timely comprehensive treatment is provided.

Treatment of ASTL

Therapy for acute stenosing laryngotracheitis is complex and depends on the stage of the disease. The treatment involves a pediatrician, otolaryngologist, resuscitator. Children with stenotic laryngotracheitis should be hospitalized regardless of the clinical form and stage of the disease. In the early days of the disease, warm drinks, warming compresses around the neck, mustards plasters on the front surface of the neck and sternum, warm socks filled with irritants (such as dry mustard) are always recommended. These measures have a positive effect on the course of the disease and may even stop it in the beginning. In addition, appoint alkaline and steam inhalation, as in acute catarrhal laryngitis.

For first-degree laryngeal stenosis, one of the pathogenetic measures is the inhalation with modern ultrasonic devices "Nebulizer" with the help of which they inhale medicines such as "Ventolin" (salbutamol), "Relenza" (zanamivir - 5 days 2 times a day): hydrocortisone at the rate of 3-5mg per 1kg of body weight or prednisolone at 1-2mg per 1kg of body weight for 2-4 days, which can be cancelled without reducing the dose. Prescribe various anti-edematous mixtures in the form of aerosols, for example: 0.5% ephedrine solution 1 ml,

0.1% adrenaline hydrochloride solution 1ml, 1% dimedrol 1ml solution, chymotrypsin 1mg in 1ml, hydrocortisone 1ml (25mg). For one inhalation 2ml of this mixture is taken 3 times a day. Other anti-inflammatory mixtures can be used. Prescribe antihistamines, tonic, sedative and vitamin therapy.

In the **second stage** of laryngeal stenosis you should increase the dose of hydrocortisone from 5 to 10mg/kg of body weight, prednisolone - up to 5mg/kg for 5-7 days. The ward should have a cool room temperature for the best functioning of the ciliated epithelium. Nowadays the hardware humidifiers are used. It is necessary to appoint dehydration and detoxification therapy at a dose of 20ml/kg, lytic mixtures to reduce the excitability of the patient. Treatment should be started already in the admission room in order not to waste

time. Spasmolytics should be introduced as soon as possible to prevent swelling (but 2% 1-2mg/kg, baralgin 0.2-0.4ml/year). With the exacerbation of croup, the use of 30 mg/kg of mulcan in 0.9% sodium chloride solution intravenously dropwise is possible. Also use antihistamines - dimedrol, fenkarol, suprastin. Diazolin cannot be used because it enhances the mucosal hyperproduction.

For stenosis of the larynx of the **third degree** perform even more intensive anti-inflammatory, dehydration and infusion therapy. The dose of hormonal drugs is increased, for example, hydrocortisone from 10 to 25mg per 1kg, prednisolone - up to 10 mg/kg, 2.4% solution of euphiline in 0.1ml/kg of body weight in children under one year, and then 1 ml per every year of a child's life. To reduce metabolic acidosis, 4% sodium bicarbonate solution is administered intravenously at 4-5mg/kg body weight. Prescribe symptomatic therapy. In the case of hyperthermia give antipyretic drugs and cool down the temperature of the child by applying cold to the projection of the main vessels. In most cases, such intensive care has a positive effect within 2-4 hours. Excitable children are recommended aminazine 0.5ml or 1ml droperidol intramuscular for children from 6 months. to 1 year, from 1 to 4 years. aminazine 1.0ml or 2ml droperidol intramuscular, older children can be given ½ tab. Glycine under the tongue up to 3 times a day. It is necessary to remember the biological role of calcium, which is the basis of bone tissue, a stimulant of nerve impulses, a universal regulator of muscle contraction, an important component of the blood clotting system. The hypocalcemic condition is noted in the genesis of laryngospasm in ASLT viral etiology. The decrease in the concentration of calcium in the blood plasma is associated with the severity of the condition in spasmodic forms. Therefore, it is rational to use calcium gluconate tablets 1g per year of life 3-4 times.

The general treatment plan includes broad-spectrum antibiotics according to indicaions. If it is a viral etiology of the disease then we use arbidol for influenza or parainfluenza, amixin IC for ARVI (Acute Respiratory Viral

Infection) (from 2 years), groprinosin 50mg/kg up to 4 times a day from the first months of life if necessary.

If the general condition worsens, conduct the toilet of the tracheobronchial tree by direct laryngoscopy by introducing into the trachea proteolytic enzymes, hormonal drugs, antibiotics of low concentration by syringe, followed by its suction along with pathological contents of the trachea and bronchi. In the dry form of stenotic laryngotracheitis with obstructive crusts, this has very positive effects. If such intensive care is ineffective, intubation is carried out with the use of general anesthesia for 3-4 days in children under 3 years of age, for 5-8 days in children of school age, and in the case of a terminal condition, a conicotomy.

The *modern method of intubation is as* follows. Under anesthesia with the use of relaxants perform a direct laryngoscopy and attentively through the mouth or nasal cavity with gently rotating movements insert special plastics thermoplastic (under the influence of body temperature become soft) tubes of appropriate sizes through the larynx into the trachea. Replacement of tubes is recommended to be carried out daily, and the baby for a while should be left without a tube to restore the local hemodynamics of larynx and trachea tissues, which prevents the formation of bedsores, granulations and scars that cause chronic laryngeal stenosis. At the same time spend intensive infusion therapy, dehydration, desensitization, symptomatic and antibiotic therapy. However, such a treatment may not have a positive effect and then it is necessary to perform a lower tracheostomy on the intubation tube.

There are upper, middle and lower tracheostomy depending on the level of incision of the trachea in relation to the isthmus of the thyroid gland. In children, it is advisable to do only a lower tracheostomy to avoid perichondritis of the larynx cartilage. The upper tracheotomy is a section of the tracheal rings above the isthmus of the thyroid gland. During the middle tracheostomy, the trachea rings are cut at the level of the isthmus of the thyroid gland. The lower

tracheostomy involves the incision of the tracheal rings below the isthmus of the thyroid gland.

If the patient's condition does not allow him to perform a lower tracheostomy, an upper tracheostomy is performed, which is technically easier to perform, especially in older children.

In an extreme situation (asphyxia), it is difficult to perform a tracheostomy, because the lack of time and appropriate conditions. In this case, a conicotomy operation is indicated in which a conical ligament is cut. It is situated between the thyroid and ring-shaped cartilages of the larynx. Various tools are offered to perform this operation, including a special tracheal tube with a tracheotomy tube, which is inserted directly through the soft tissues of the conical ligament. However, these tools are virtually non-available now. Therefore, the operation is performed as follows. The patient's position is horizontal. Palpation determines the location of the soft tissue incision. Under local anesthesia (0.5% novocaine solution), and sometimes without it, the skin is cut transversely; then bluntly find a conical ligament that is yellow. It is also cut by a cross-section into which a tracheotomy cannula or tube of appropriate size is inserted. It should be noted that conicotomy is a temporary intervention on the larynx. After restoration of breathing, you should immediately do an upper tracheostomy according to all rules, and the suture of the soft tissue in the area of the conicotomy must to be sewed. Sometimes a conicotricotomy, a cricotomy and even a thyrotomy instead of a conicotomy. After these operations, further tactics are the same as for conicotomy.

In the postoperative period, it is necessary to prescribe antibiotics and other anti-inflammatory therapy for the prevention of laryngeal perichondritis. The skin around the tracheostoma is greased with zinc ointment or Lassar paste. The tracheostoma should be treated as a clean wound. Make sterile dressings daily, change the tracheotomy tube. In the postoperative period, the mucous membrane of the cannula may become obstructed, causing respiratory failure. Therefore, the inner tube of the cannula should be periodically removed and

thoroughly cleaned with a cotton swab wound around a metal probe, treat with alcohol and reinsert into the cannula. If this is not enough to eliminate respiratory failure, the airways are flushed and the contents below the tracheotomy tube must be aspirated, as crusts, mucus and blood clots can accumulate there. In the case of ineffective manipulation, it is necessary to remove the entire tube and with the help of a significant nasal dilator, and better dilator Killian, examine the lumen of the trachea and bronchi. Thick mucus, blood clots and crusts are removed with forceps or cotton wool wound on a probe. It is recommended to pour 5-6 drops of warm isotonic sodium chloride solution into the tracheotomy tube every 2-3 hours. Hydrocortisone and proteolytic enzymes can be added to the isotonic solution. But it is not advisable to use the latter ones for a long time. Applying different types of oil, such as peach or sea-buckthorn, petroleum jelly, fish oil is also inappropriate and even harmful. The sutures are removed for 6-7 days.

To restore the drainage function of the lower respiratory tract, it is necessary to remove sputum. To do this, create a microclimate in the ward with high humidity, periodically suck sputum through the tracheotomy tube. Therefore, the diameter of the suction cup tip should be smaller than the lumen of the cannula. The tips are stored in antiseptic solutions. Before suctioning the sputum it is necessary to make a massage of the chest, in the tracheostomy pour an isotonic sodium chloride solution from the syringe (not pipette) until the cough reflex appears. The suction procedure is repeated until the pathological contents are completely removed. Suction frequency depends on the activity of the inflammatory process in the trachea and bronchi, as well as on the condition of the child.

In the postoperative period, it is necessary to apply therapeutic and respiratory exercises, chest massage and nutrition. It is necessary to create sanitary and hygienic conditions in the wards where such patients are in. All precautionary measures should be taken. It depends on the etiological factor and the technique of surgery, as well as the general condition of the body.

Prolonged cannulosity leads to chronic scarring of the trachea, chronic bronchitis and pneumonia.

Before decanulation, it is necessary to conduct a clinical examination of the patient, as well as direct laryngoscopy, bronchoscopy, according to the indications - radiological examination, to make sure that there are no obstacles to decanulation, if any, they are eliminated. It is advisable to perform decanulation in such a way as to insert into the tracheostomy tubes of smaller size up to No. 00 and No. 000 daily, closing their lumen during the day. At night, the tracheotomy tube does not close. The decanulation is carried out in the morning, the child is looked after individually and in the event of respiratory failure of varying degrees, the tracheotomy tube is introduced into the trachea.

Treatment of diphtheria croup in children

After the diagnosis of diphtheria, in the first 2 hours after hospitalization, the antitoxic antidyphtheria serum must be administered.

Table 11

Dosage of anti-diphtheria serum in diphtheria croup (thousand IU)

Clinical form	Localized croup	Common croup	Descending croup
1st dose	30-40	----------	30-40
Repeated dose	40-50	20-30	60-80
Total dosage	40-50	20-30	60-80

Control questions:

- Perform differential diagnosis between ASLT and diphtheria.
- What are the etiologic factors of ASLT.
- What is the method of incubation of the trachea?
- What is the intensive care for ASLT 3 stage in a child of 1 year?
- Remember the essence of the method of administration of foreign sera.

EDEMA AND SWELLING OF THE CEREBRUM SYNDROME.

Cerebral edema-swelling is a clinical syndrome due to brain tissue swelling with increased volume and increased cranial pressure. This syndrome is divided into two stages: swelling - the accumulation of bound water in cells; edema - the accumulation of free water in the intercellular space. Swelling of the brain - is a reactive dynamic process caused by damage to the blood-brain barrier (BBB), disorders of cerebral circulation with secondary metabolic processes, water-electrolyte metabolism and accumulation of water in extracellular and intracellular brain spaces, impaired nerve function. In the clinic of infectious diseases BES (brain edema-swelling) occurs most often in course of inflammatory processes in the brain and meninges: bacterial and viral meningitis, encephalitis; diseases accompanied by the development of cerebral vasculitis (severe forms of influenza, hemorrhagic fever, malaria).

Pathogenesis. The concept of "swelling-edema" corresponds to the development of two processes - direct swelling with the accumulation of fluid in the intercellular spaces of the brain and swelling of cells as a result of the accumulation of sodium in them, which leads to an increase in the volume of intracellular fluid. The edema is localized mainly in the white and the swelling in the gray matter of the brain.

The morphological substrate of BES is:

- Damage to the endothelium of the vessels of the brain;
- Increasing the permeability of the vessel wall;
- Release of peripheral catecholamines and hyperlactocidemia;
- Blood stasis and microthrombosis of capillaries;
- Brain hypoxia;
- Diapedetic hemorrhage;
- Hyperhydration of pericellular and pericapillary spaces (brain swelling);
- Increase in liquor production;
- Diffuse swelling of neurocytes and glial cells (brain swelling);

• Formation of hypertensive syndrome, development of complications (hydrocephalus, inclination of the medulla oblongata into the large occipital opening, Waterhouse-Friederichsen syndrome).

Symptoms: convulsions, initially clonic, then tonic. Cramps of the decerebral type, hyperthermia (40 ^{0}C and above), shortness of breath, marbling of the skin, tachycardia, poor filling of the pulse, drop in blood pressure, enlargement of the liver and spleen, pastosity of the feet and legs, paresis of the intestine; Disturbance of consciousness - somnolence, sopor (lack of linguistic contact while maintaining sensitivity and reflex sphere), coma (suppression of cortical functions with no reactions to external stimuli, movement disorders, sensitivity, reflexes).

In I degree coma, muscle tone decreases, skin and tendon reflexes are partially suppressed, pain response is only positive to strong and deep pain stimuli. Coma of II degree is characterized by narrow pupils, sluggish reaction to light, muscular hypotension, lack of sensitivity, COMPLETE fading of skin and tendon reflexes, partial suppression of swallowing and cough reflex. In grade coma, the pupils are wide, with no reaction to light, muscle hypotension, complete reflexion. There is an injury of the respiratory center - respiratory arrhythmia: uniform in amplitude respiratory movements with periodic pauses (Biot breathing), subsequently - periodic inhalation with increasing and decreasing amplitude movements (Cheyne-Stokes), then - agonal breathing in breathing (Kusmaul). Lesions of the cardiovascular center: arterial hypotension and the development of circulatory collapse.

Diseases that can cause BES: meningitis, encephalitis, meningoencephalitis, common infectious diseases with toxicosis, etc.

Procedure

1.Prophylaxis and therapy for respiratory disorders:

• In case of primary lesion - as a result of violation of the respiratory center (respiratory arrest, bradypnoe, pathological type of breathing) - indicate the patient to artificial lung ventilation;

• Secondary lesions caused by obstruction of the upper and lower respiratory tract, secondary respiratory tract infections, convulsions, hyperthermia - toilet of the upper respiratory tract (suction of mucus, phlegm, vomiting), replacement of the tongue, lower jaw, fix the headposition to provide the maximum passability of air passageways, in the absence of effect - tracheal intubation;

• Lower respiratory tract: cough stimulation by suction catheter; microintubation (catheter insertion through the nose into the trachea); microtracheostomy (percutaneous puncture and catheterization of the trachea), toilet intubation of the trachea, bronchoscopy, in the absence of effect - tracheostomy;

• In the case of pronounced salivation - atropine - 0,1% solution - 0,01-0,03ml/kg - single dose intravenously;

2. Correction of disturbances of function of cardiovascular system:

• In tachycardia (often with ventricular extrasystole) - eliminate factors that contribute to increased cardiac activity (respiratory failure, hyperthermia, convulsions, hypovolemia, acid-base disorders), after which you can apply drugs that reduce the myocardial excitability 2: - 0.05 - 0.1ml/kg - single dose intravenously slowly (10-15 min) on saline or 5% glucose solution, repeating this dose after 20-30 minutes to a total dose of 0.25ml/kg; or aminazine intravenously by pharmacological effect (1ml of 2.5% aminazine solution is diluted in 9ml of saline and injected 0.5-1ml of the solution; if pressure does not decrease, it can be re-injected every 5-10 minutes);

3. Correction of disorders of water-electrolyte metabolism and dehydration therapy:

• Plasma - 10-15ml/kg - single dose intravenously; albumin 10-20% solution - 10-15ml/kg - single dose intravenously; lasix - 2-4mg/kg single dose intravenously; mannitol 15-20% solution - 1-2g/kg - single dose (5-10ml/kg - single dose) intravenously, or sorbitol 20% solution - 1-1,5g/kg - single dose (5-7,5ml/kg - single dose) intravenously; Reogluman - 10-15ml/kg - single

dose intravenously; eufilin 2.4% solution - 0.1ml/kg - single dose - intravenously slowly in saline every 6-8 hours; - glucose 10-20% solution – 20 ml/kg/single dose with insulin (1U per 4g glucose), potassium chloride – 7, 5% solution - 1ml/kg / single dose, cocarboxylase - 8mg/kg/single dose, trental - 5mg/kg/single dose, intravenously. Glycerin - 1ml/kg/single dose in the stomach.

For the first 2-3 days of dehydration, the water balance becomes negative in general by 20-25ml/kg of body weight. In other words, for this day, more fluid is released from the body than is injected in an amount of about 5% of body weight. In the next day, the water balance is already kept at zero - how much the body loses fluid, as much as it is introduced, that is, the child is kept on a dehydration regime with water deficiency in the body in the amount of about 5% of body weight (to prevent increased hematocrit above 44-46%). The dehydration regimen is kept for about 7-10 days, and subsequently the water deficiency (5% of body weight), which was reached in the first 2-3 days, gradually fills up within 2-3 days.

4. Correction of acid-base disorders (CBS):

Establishment of effective alveolar lung ventilation (elimination of airway obstruction, artificial lung ventilation). Sodium bicarbonate solution 4% - 3-6ml/kg/ single dose intravenously (carefully!) Or trisamine 0.3% solution - 12,5ml/kg/ single dose intravenously, under the control of urine pH litmus indicator tape, seeking increase urine pH to 6.5-7.0. Early elimination of hyperthermic and convulsive syndromes is the most common cause of metabolic acidosis.

5. Elimination of disorders of the gastrointestinal tract:

• Introduction of a permanent probe for gastric drainage, vomiting and regurgitation followed by aspiration into the respiratory tract; proserin 0.05% solution - 0.1ml per year of life / single dose (but not more than 0.75ml) - subcutaneously and intravenously 1-2 times a day, or galantamine 0.25% solution - 0.1ml/ per year of life / single dose - subcutaneously and

intravenously - 1-2 times a day, combined with atropine (or other cholinolytics), if necessary - with droperidol, aminazine, pentamine - without reducing blood pressure. Elimination of intestinal swelling.

6. Hormonal, anti-inflammatory therapy:

• Dexamethasone (the drug of choice because it does not cause sodium and water retention in the body) - 0.5mg/kg/ per day, divided evenly into 4-6 injections intravenously or intramuscularly; or prednisolone - 3-5mg/kg/ day, divided evenly into 4-6 injections intravenously or intramuscularly; or hydrocortisone - 15mg/kg/ day, divided evenly into 4-6 intravenously or intramuscularly.

N. B. Dexamethasone is 7 times more active than prednisone, 30 times more active than hydrocortisone, 35 times more active than cortisone.

Some authors recommend the combination of these drugs, so they have varying degrees of influence on the inflammatory process, carbohydrate and mineral metabolism. The course of corticosteroid therapy in severe brain edema lasts no more than 1-2 weeks, depending on the subsequent clinical course of the disease, then the dosage of corticosteroids in each subsequent day is reduced twice and reaching a dose of 0.05-0.1mg/kg/day (for prednisolone), completely canceled.

• The use of barbiturates (their ability to reduce swelling, reduce intracranial pressure, prevent and reduce convulsive activity, promote stabilization of brain lipid membrane cells, reduce brain O2 consumption) - scheme: thiopental-10% solution / kg 10% solution / kg single dose (0.1ml/kg) every three hours intramuscularly at a daily dose of 80mg/kg (0.8ml/kg). If the convulsive syndrome is not completely stopped, a single dose of 10mg/kg is given every 2 hours, i.e. the child receives 120mg/kg (1.2ml/kg). This therapy requires constant medical observation of the child (maintain airways passagibility, monitor respiratory parameters, cardiovascular system activity, CBS and blood gas composition). The authors recommend thiopental sodium therapy to be

started in the early stages of edema and to perform it no more than 4-6 days depending on the condition of the child.

• Desensitizing therapy (antihistamines - H1-histaminolytics): dimedrol 1% solution - 0.2-0.3ml/kg/day, divided into 3 intravenous, intramuscular injections; or diprazine (pipolphene) 2.5% solution - 0.1ml/kg/day, divided into 3 intravenous, intramuscular injections; or suprastin 2% solution - 0.1-0.15 ml/kg/day, divided into 3 intravenous, intramuscular injections. When using these drugs in combination with sedatives and anticonvulsants, it is necessary to monitor the baby's breathing very carefully.

• Proteolytic enzyme inhibitor therapy:

contrical (tracilol, tsalol) - 1000-2000U/kg/ daily dose, divided into 2-3 injections, intravenously (slowly!) or dropwise into 5% glucose solution or saline; or Gordox - 10000 – 20000U/kg/ daily dose, divided into 2-3 injections, intravenously dropwise into 5% glucose solution or saline. It is recommended to determine individual sensitivity using a skin sample (0.2 ml injection).

10. Antibacterial therapy: for the purpose of prevention of secondary infection (more often hospital, that is, low-sensitive) - broad-spectrum antibiotics - aminoglycosides II - III generation, cephalosporins III-IV generation, semi-synthetic penicillins, carbapenems, monobactins, levomitochechin, levomitsocin. When developing a secondary infection or purulent meningitis (meningoencephalitis) after receiving the results of determining the sensitivity of the microflora to antibiotics – begin to introduce of the appropriate drug.

In anaerobic infection - metronidazole - 7.5mg/kg/single dose - intravenously dropwise 3 times a day; in combination with lincomycin – 10-20mg/kg/daily dose - divided into 3-4 intramuscular injections; or clindamycin - 30 - 40mg/kg/day - divided into 3-4 intramuscular injections. Lincomycin and clindamycin can be administered intravenously, but slowly (dropwise!) - not less than 20 minutes - prevention of neuromuscular blockade, impaired function of the cardiovascular system. Antibiotic therapy should be performed in combination with antifungal drugs and B vitamins.

• General care: timely feeding (by mouth, probe); changing bed; keep to aseptic and antiseptic care; care of the intubation tube; establishment of effective humidification of the inhaled gases; bedsores prevention, baths, body rubs.

Control questions:

- What is the mechanism of development of edema-swelling of the brain? What is the clinical picture of BES?
- Design a plan of urgent measures at BES.

TOXIC SHOCK SYNDROME (TSS)

In severe forms of infectious diseases, the most serious complication of septic condition and bacteremia - infectious-toxic shock, may occur. The cause of TSS is the direct action of the infectious agent, the immaturity of the baby's epithelial barrier, the violation of detoxification and the elimination of the microbe, which leads to bacterial translocation and a cascade of inflammatory, biochemical, immunological reactions.

The main pathogenetic mechanisms of TSS:

- damage to cells by toxins;
- adrenal activation, catecholamine excretion;
- Central nervous system (CNS) stimulation (inhibition);
- spasm of vessels, reduction of capillary tissue blood flow;
- hypoxia of organs and tissues;
- accumulation of under-oxidized products, cellular acidosis, increase of lactic acid;
- blood stasis in the capillaries, relative hypovolemia;
- metabolic acidosis;
- release of histamine, serotonin and other biologically active substances;

cellular hyperhydration, outflow of fluid from the intravascular channel (absolute hypovolemia);

- aggregation of blood cells, microthrombosis of vessels, block of microcirculation;
- consumption coagulopathy;
- degenerative changes in organs and tissues;
- brain swelling, lungs, death.

TSS can also be exacerbated *by endogenous metabolic intoxication* - an endotoxinemia of gastrointestinal origin caused by both bacteria and viruses.

Classification

I degree shock (compensated shock), characterized by the following signs: severe general condition, hyperesthesia, anxiety, arousal, anxiety, pallor, acrocyanosis, tachycardia, blood pressure (BP) within standard, moderate shortness of breath, decrease in diuresis. Algover's index (the ratio of the pulse rate to the maximum BP), which allows us to estimate the deficiency of blood volume, increases to 1.0 (N = 0.5-0.6)

II degree shock (subcompensated shock) - is characterized by inhibition, confusion, skin pallor, general cyanosis, tachycardia, hypothermia, hypotension (blood pressure within 80/60 - 60/20 mm Hg), shortness of breath, oliguria, signs of DVZ syndrome. Hypoxemia, hypokalemia, decompensated acidosis are determined. Algover's score is 1.0-1.5

III degree shock (decompensated shock) - is manifested by a violation of consciousness resistance and coma, general cyanosis, severe dyspnoe, hypothermia (lowering the temperature to subnormal values), pulse is filiform or absent, hypotension (AO 50/0. Unavailable for determining), anuria, decompensated metabolic acidosis, deep hypoxia, signs of DIC, irreversible changes in the internal organs. Algover's score is over 1.5. With the further development of toxicosis passes into a stage of **coma**: **midbrain**, then stem (**bulbar**), and then **terminal**. Consciousness is lost, muscular hypotonia develops, motor activity disappears, and clonic-tonic convulsions often appear. Peripheral blood flow deficiency is progressively increasing: skin is gray-cyanotic, hemorrhagic elements (result of DIC syndrome), "hypostasis" (by the type of cadaver stains) can be observed. BP decreases, heart tones are quite, tachycardia is changed by bradycardia (a prognostically negative sign, indicating swelling and swelling of the brain), tachypnea changes to bradypnoe, respiration superficial, periodic. Vomiting "coffee grounds," paresis of the intestines and sphincters. A complete reflexion develops. In the lungs, a swelling pattern, pink foam on the lips. The skin cyanosis grows hypothermia, the skin becomes cold, covered with acidic, sticky sweat. The terminal phase (coma) is characterized by

complete reflexion, cessation of vessels, muscle hypotension, disappearance of the swallowing reflex, depression, and then respiratory and cardiac arrest.

Precursors of TSS development:

TSS syndrome develops very rapidly, sometimes even unexpectedly. The deterioration occurs on the background of a steady increase in body temperature above 39 ° C - hyperthermic syndrome. Characteristic is the excitation of the child, anxiety, tremor of the hands, swelling and tension of the big vertex, rigidity of the occipital muscles, with increasing severity - generalized clonic-tonic convulsions.

Criteria for the diagnosis of TSS:

- High temperature, the pronounced toxicosis in the onset of the disease;
- Tachycardia, which does not correspond the level of fever, in the future - the presence of tachycardia on the background of normalization of body temperature;
- Reduction of blood pressure;
- The appearance of acrocyanosis, dyspnoe, oliguria;
- Development of DIC and other manifestations of multiple organ failure syndrome.

Intensive care TSS

Pre-hospital stage of treatment: the doctor's tactics include providing emergency care for the patient at the site of occurrence, ensuring his immediate transportation and hospitalization to the specialized hospital. Before transportation and at during it, therapeutic measures must be carried out, the purpose of which is to support the vital functions of the patient's body on the way to the hospital. In this case, it is always should be taken into account the preliminary etiological diagnosis for the appointment, in some cases, of etiotropic therapy at the pre-hospital stage

- Provision of venous access.
- Antibacterial therapy - levomycetin sodium succinate at 25mg/kg (single dose) intravenously

• Glucocorticoids - prednisolone, hydrocortisone or dexazone 2mg/kg on prednisolone - without TTS, 5mg/kg - with TTS degree I, 10mg/kg - with TTS degree II, 15-20 mg/kg - with ITS III degree.

• Infusion therapy with saline solutions or reopolyglukin to stabilize BCC.

• Inotropes (dopamine) - to support hemodynamics.

• Depending on the severity of the hospitalization to the intensive care unit or the department of neuroinfection of the infectious hospital.

• Antibacterial therapy: in the presence of TSS - third-generation cephalosporins - cefatoxime 100-200mg/kg/day, ceftriaxone IO mg / kg / day, levomitsetin succinate at a dose of 100mg/kg day, after termination of TSS 200 appoint penicillin. Units / kg / day. In severe form and the need for protection against nosocomial infection, additionally used 3rd generation aminoglycosides - amikacin up to 20mg/kg/day, netilmicin 1.5-2mg/kg every 8 hours.

• Detoxification therapy in moderate forms is carried out by glucose-saline solutions taking into account the daily need for fluid and pathological costs.

• Post-syndromic therapy is carried out in accordance with the existing syndromes, their treatment is carried out according to the appropriate treatment protocols.

General principles of intensive care.

The basis of TSS treatment is the restoration of circulating blood volume, microcirculation, prevention and relief of DIC, achieved through intensive infusion therapy and the introduction of pharmacological drugs.

• As infusion solutions use crystalloids (glucose-insulin-potassium mixture), colloids, (reosorbylact, albumin) in a ratio of 3: 1. On the background of hemodynamic stabilization, it is advisable to use 400-500ml of rheogluman intravenously, which together with the improvement of rheological properties of blood stimulates diuresis, promotes the relief of renal failure. In order to reduce metabolic acidosis, 300-400ml of 4% sodium bicarbonate solution is

administered. The total volume of infusion solutions is up to 50ml/kg per day, the injection rate at low blood pressure is 500 ml/h.

• For correction of microvascular disorders in the vital organs, delay in the development of DIC, anti-inflammatory action, stabilization of lysosomal membranes prescribe ACS in doses equivalent to 10-15mg of prednisolone per 1kg of body weight of the child, depending on age. With grade III TSS, the prednisolone dose can reach 30mg/kg/ day (or 3mg/kg/day of dexamethasone). ACS is prescribed for a relatively short time -24-48 h with subsequent dose reduction and avoidance of the drug after the shock termination.

• Dopamine is used at average doses of 3-10mcg/kg for 1 hour to stabilize hemodynamics (IIS-II degree IIS).

• Heparin is an important tool in the treatment of TSS. It is administered intravenously first full dosage, then dropwise - 5 thousand units under the control of the time of blood coagulation (not more than 18 minutes). The daily dose is 500U/kg, in the stage of hypocoagulation - 100-250 IU / kg.

• At IIS-III degree TSS it is obligatory to introduce fibrinolysis-protease inhibitors (contrical 1000U/kg or Gordox 7000 U/kg) intravenously dropwise intravenously on glucose or saline sodium chloride.

• Constant artificial lung ventilation with moist oxygen (respiratory volume 6-8 ml/kg of body weight, but not more than 12 ml/kg of body weight).

• Etiotropic therapy of the main disease is carried out simultaneously with resuscitation measures, its appointment at the earliest possible time is important. Termination of infusion therapy is possible after 6h of satisfactory hemodynamics and restoration of diuresis. The use of vasopressor active agents (adrenaline, noradrenaline, mesaton) is contraindicated due to the risk of developing severe vasoconstriction. The introduction of these drugs is acceptable only in the absence of the effect of dopamine infusion and the above mentioned measures.

Control questions:

- What are the main pathogenetic mechanisms of TSS?
- Describe TSS by its severity.
- What are the precursors of TSS?
- List the intensive care measures for TSS.
- What is your prognosis for TSS?

WHOOPING COUGH.
INTENSIVE THERAPY OF RESPIRATORY ARREST AND PECULIARITIES OF THERAPY.

Whooping cough is an acute infectious disease caused by the Bordeaux-Zhang bacterium and characterized by a cough with repeats and possible respiratory arrest (sometimes up to 10 times a day). The most severely ill are newborn and infants.

Severity criteria:

- the severity of symptoms of oxygen deficiency beyond coughing attacks;
- frequency and nature of convulsive cough attacks;
- vomiting after a convulsive cough;
- the condition of the child in the period between attacks;
- presence of specific and nonspecific complications;
- severity of hematological changes.

The whooping cough clinic in children

The incubation period of pertussis lasts from 3 to 15 days, with an average of 5-8 days. The disease can be divided into three periods.

The catarrhal period is characterized by a moderate increase in temperature: sometimes the temperature is subfebrile or even remains normal, a significant increase in body temperature (up to 39° C and above) is rarely observed. From the first days of the disease onset there is a dry cough with no specific features. Gradually this symptom increases, becoming the main one in the picture of the disease. Already at the end of the catarrhal period, the cough acquires the nature of more or less prolonged attacks and has two peculiarities: it occurs mainly at night and ends with vomiting. Often in the catarrhal period there is a runny nose. The patient's well-being either does not change, or slightly worsens, the appetite remains. The catarrhal period lasts 3-14 days. Sometimes, especially in infants, it is reduced to 5-7 days, sometimes, on the contrary, it can be prolonged.

The transition to the second spasmodic period is gradual. Typical attacks of spasmodic or convulsive cough occur suddenly or after short precursors: a sore throat, a squeeze in the chest and anxiety. The attack consists of a series of short coughing pushes that go one after the other without a pause. Then the patient takes a deep convulsive breath, which due to the spastic narrowing of the glottis is accompanied by a whistling sound (reprise). After that, the attack continues in the form of the same coughing pushes with subsequent wheezing. There may be several reprints during a cough attack. The heavier the form of whooping cough, the longer the cough attacks and the greater the number of reprises they are accompanied. The cough attack ends with a cough of viscous transparent sputum, sometimes vomiting. In severe attacks, cough sputum can have impurities of blood. Vomiting after a seizure is not an entirely constant sign. The heavier the form of whooping cough, the more often it is observed. With a mild form of whooping cough, vomiting is rare or absent.

During a cough attack, the patient has a very characteristic appearance: the face is red or even blue, the cervical veins are swollen, the eyes are filled with blood, the tear appears, the tongue extends outwards, its tip is bent upwards. Spontaneous urination and defecation are possible during a severe attack. High tension can lead to conjunctival hemorrhage, nasal bleeding, and development of cerebral circulation disorders. In severe coughing attacks, respiratory arrest may occur. Cough attacks are caused by various external stimuli (throat examination, dressing and undressing, feeding, loud noise, crying of children, etc.). Many clinicians have noted that cough attacks occur mainly at night. In the daytime, especially when walking in the open air, the baby coughs much less often or does not cough at all. The convulsive cough reaches its maximum at the end of the second week, then gradually disappears. Frequent coughing attacks accompanied by circulatory disorders, the patient's face becomes puffy, eyelids swell, hemorrhages often appear on the skin and conjunctiva. Swelling can occur not only on the face but also on the whole body (in severe cases), especially on the lower extremities. On examination of the oral

cavity, on frenum of the tongue sometimes found a wound that is later covered by a white plaque in the form of growth. This wound is the result of mechanical friction of the frenum and the sharp edges of the lower incisors. When the whooping cough subsides, the wound gradually decreases and disappears. Even with frequent cough attacks with uncomplicated whooping cough, the general condition of most patients does not worsen. Children with whooping cough, in the interval between cough attacks have a normal lifestyle, play, have good appetite. Body temperature, slightly elevated in the catarrhal period, by the time of the onset of cough attacks in most patients decreases to normal and only sometimes is subfebrile. A clear fever in the spasmodic period, as a rule, indicates the presence of some complication. Only in some patients with uncomplicated whooping cough fever persists for a long time. During the study of the lungs often found signs of emphysema, tympanic or box tone in percussion. Auscultation is determined by dry or wet rales. Radiographically revealed an increase in the transparency of the pulmonary fields, low standing and flattening of the diaphragm, an increase in the shade of both guillotines, the strengthening of the mesh pulmonary pattern, the appearance of linear weights. With the further course of the disease, mainly in the 5-7th week, intense tendencies are formed, coming out of the gill and extending mostly down to the diaphragm. Sometimes these strands form a triangular figure with a vertex near the ridge, approximately at the level of the gillus, and a base on the diaphragm. These radiological changes are gradually disappearing at the junction stage. From the cardiovascular system note the acceleration of the pulse during the onset of coughing, increased blood and venous pressure. There is a decrease in the resistance of the capillaries, which causes bleeding into the skin and mucous membranes. In severe whooping cough, the heart is covered by emphysematous lungs or significantly enlarged by the right ventricle. On a. pulmonalis sometimes heard the accent of the second tone. On the part of the nervous system, the patient's irritability is observed, in severe cases - flaccidity, adynamia, sleep disturbances, convulsive mimic muscles, occasional dullness of

consciousness. During the blood test, most patients show significant leukocytosis and lymphocytosis. The number of leukocytes can reach 20-70 thousand or more. The degree of leukocytosis depends on the severity of the disease. ESR is reduced or normal. These hematologic changes are already observed in the catarrhal stage and disappeared with the elimination of whooping cough infectious process. In patients who have previously been vaccinated against whooping cough, changes in the cellular composition of the blood are less common, their severity is low. The spasmodic period lasts from 2 to 8 weeks. Gradually, the frequency of attacks decreases, their strength weakens, the disease goes into the third period. During convalescence, the cough loses convulsiveness and becomes less frequent. The phlegm becomes mucous-purulent. Gradually all symptoms of the disease disappear. This period lasts 2-4 weeks. Therefore, the total duration of the disease ranges from 5 to 12 weeks. Sometimes the process is continued for a longer period. At the stage of convalescence, or even after complete elimination of all symptoms of whooping cough, typical cough attacks are sometimes returned - these are false repeats. It occurs after the organism is healed from whooping cough and are not accompanied by a typical whooping cough reaction from the blood. These "repeats" occur in patients during recovery in case of concomitance of any infectious disease (influenza, tonsillitis, measles, etc.). There are three main forms of whooping cough: mild, moderate and severe. In mild form, the frequency of attacks reaches up to 15 per day, the number of reprisals - up to 5; the attacks are typical but brief; vomiting is observed relatively rarely, the general well-being of the patient does not deteriorate. In moderate form, the number of cough attacks reaches up to 25 per day (each of which is prolonged), the number of reprisals - 5-10; often vomiting occurs at the end of attacks. Overall well self-feeling, but moderate. In the severe form of whooping cough, there are about 30-50 or more cough attacks per day; attacks are severe and sometimes last up to 15 minutes, have more than 10 repetitions and almost always end in vomiting; sleep disturbances, lack of appetite, lethargy, weight

loss, and often fever are observed. The criteria for the severity of whooping cough by the number of attacks proposed by Filatov N.F. are fairly conditional. Yes, even if the infant has a moderate incidence of short-term cough attacks, whooping cough can have very difficult course. Recently, the erased form of whooping cough has been increasingly appeared, characterized by the absence of typical coughing attacks with reprisals and a shortened course. In these cases, tracheitis or tracheobronchitis are often diagnosed. Such forms are more commonly seen in vaccinated children. There is also an asymptomatic form of whooping cough in which clinical manifestations are absent, although cyclical immunological, sometimes hematologic changes, radiological changes, pulmonary blood flow, changes of the capillary system occur in the body. Studies of cerebral hemodynamics in whooping cough indicate an increase in peripheral resistance of brain vessels, a decrease in systolic and diastolic slowing blood flow in the brain, which increases its hypoxia. Changes of cerebral hemodynamics at a moderate form of whooping cough are unchanged for 3-4 months, and in severe forms - up to 1 year. In children who have been vaccinated against whooping cough, the disease tends to be mild and in erased form compared to unvaccinated ones, with less pronounced hematologic abnormalities, complications occur less frequently, the course and prognosis of the disease more favorable.

Features of whooping cough in children of the first year of life

In infants, whooping cough has a number of features. There is a reduction in incubation (up to 3-5 days) and catarrhal (up to 2-6 days) periods; sometimes the catarrhal period "falls out", and convulsive cough is noted from the first days of the disease. Cough attacks in most infants are not accompanied by reprisals. Vomiting, hemorrhagic symptoms and edema are less common than in older children. Cough attacks often lead to apnea. The disorder of gas exchange is more pronounced than in children of preschool age, and cyanosis is more often observed. Young children are especially sensitive to oxygen deficiency: hypoxia complicates the course of the process, contributes to the development of

complications. In infants more often than in older children, there is a dullness of consciousness, seizures of epilepticform seizures, seizures of mimic muscles. The whooping cough of children under 6 months is especially difficult. Due to the lack of teeth, the formation of wounds on the frenum of the tongue in children aged 6-8 months is very rare. The duration of the spasmodic period can increase up to 2-3 months. More often than not in older children, respiratory complications: bronchitis, bronchopneumonia are identified. Pneumonia in infants is characterized by early development, mostly of draining nature, prolonged, and have high lethality - it is the leading cause of death from whooping cough.

Features of a modern whooping cough clinics.

In the last 20-30 years, the whooping cough clinics has undergone significant changes. The share of light and erased forms has increased. The frequency of complications and the mortality rate decreased sharply. However, among children under the age of 1 year, especially under 6 months who have not undergone or have not completed active immunization, whooping cough remains a serious illness and is often the cause of death. The "relief" of the whooping cough clinics is primarily caused by the mass preventive vaccinations. Changes in the biological properties of the pathogen may also have some significance. The Bordetella whooping cogh has recently changed: circulating serotype 1, 2, 3 has recently changed to the less virulent serotype 1, 2, 3.

Diagnosis of whooping cough. The most important condition for effective control of whooping cough is its early diagnosis in the catarrhal stage, when the patient is most contagious. However, the diagnosis of whooping cough in the catarrhal period has many difficulties, especially in the case of atypical disease and in children under 6 months. In the case of the diagnosis of whooping cough we should take into account the specific features of the clinical course (cyclicity, paroxysmal cough with reprisals, viscous phlegm and vomiting at the end of the cough attack, the typical appearance of the patient, a wound on the

frenum of the tongue, etc.). Also important are typical hematologic changes (lymphocytic leukocytosis with reduced or normal ESR, which may persist for up to 5 weeks from the onset of the disease), radiographic data (presence of "pertussis triangles" - segmental or polysegmental atelectasis in the lungs). Of great importance is the epidemiological history: contact with a patient with a typical whooping cough or with a person who has been coughing for a long time (atypical whooping cough).

Assistance in the diagnosis of whooping cough, especially in its early stages, is provided by a bacteriological method. During the study of whooping cough, collect material from the nasopharynx with a sterile cotton swab with a bent end, so that the material can be removed from the walls of the pharynx and from under the tongue. After cultivation bacterioscopy is performed and the culture and agglutinating properties of suspected colonies with specific sera are studied. The microbiological method is of great value for the diagnosis of whooping cough. It should be noted that in the case of antibiotic treatment, the ability to inoculate whooping cough is dramatically reduced. For the purpose of accelerated diagnosis, an immunofluorescence method, a PCR method, can be used by which the whooping cough microbe can be detected directly in smears of nasal mucosa.

Treatment of pertussis in children

The most important role in the treatment of patients with whooping cough is played by a properly organized regimen and supervision of the patient. Bed regimen is prescribed only in the case of fever and complications. Children of the first year of life are subject to compulsory hospitalization, as skilled care is very important for them. It is recommended to keep ill infants with severe whooping cough in a darkened quiet room, as rarely as possible disturb them, as exposure to external stimuli may cause severe paroxysm of cough with apnea. Fresh, cool, moist air is very good for patients with whooping cough. Prolonged stay of the patient in the open air improves ventilation, oxygen exchange and may have a reflexive effect on the CNS. In this case, cough attacks become rarer

and weaker. The child should spend most of the day outdoors in the summer, and several hours a day in the cold months of the year. In winter, walks should take place in covered accommodations. It is allowed to walk for patients at an air temperature not lower than -10°C. Of course, we should not allow the heat loss of the child, and the individual sensitivity to such walks should be taken into account. Constant thorough ventilation of the patient's room should also be ensured. Much attention should be paid to the educational work with older children: organizing their leisure, various activities, games and more. Children who are passionate about playing are less likely to cough. Emotional and physical stimuli that can trigger coughs should be eliminated. Food of the patient with whooping cough is cooked taking into account the vomiting, possible after coughing, it seriously complicates the consumption of food. High-calories, high-grade, concentrated, semi-liquid, vitamin-rich foods are recommended. Feeding of the patients should be in small portions after a cough attack. After feeding, it is necessary to protect the baby especially from the influence of stimuli provoking the development of cough attacks (various diagnostic and medical manipulations, examination of the pharynx, etc.). If vomiting occurs shortly after feeding, the latter should be repeated. Very frequent vomiting requires parenteral administration of fluid. In the treatment of patients with whooping cough as a specific (etiotropic) treatment we use antibiotics - erythromycin, ampicillin. Antibiotics are indicated as starting drugs for the probable diagnosis of whooping cough or to prevent its spread. The administration of antibiotics in the spasmodic period of the disease will not affect the course of the disease, but will help to release the child from the whooping cough and prevent the spread of infection in the environment. The first-line antibiotic in whooping cough patients is erythromycin at a dose of 50mg/kg of body weight (no more than 2g per day), reserve drugs - ampicillin at a dose of 100mg/kg of body weight per day, co-trimoxazole at a dose of 8mg/kg of body weight by trimethoprim or 40mg/kg of body weight per day for sulfamethoxazole. The course of antibacterial therapy for whooping cough lasts 14 days. The main goal of treating severe forms of

whooping cough is to cure hypoxia that develops as a result of reducing the flow of oxygen through the respiratory tract during cough attacks. The first step in resolving this issue should be the prevention of new attacks of coughing with the help of security mode: to eliminate all external emotional stimuli, if possible - intramuscular injections, physiotherapy, there should be no bright light in the hospital room, loud sounds, constant ventilation of the wards, in severe frequent attacks coughing patient is transfered in an oxygen tent. A 2.5% solution of chlorpromazine, 1-2.5mg/kg of body weight, is administered intramuscularly twice a day, before daytime and nighttime, with medicines to prevent and reduce cough attacks. In children of the first year of life, preference is given to a titrated solution of chlorpromazine, which is prepared at the rate of 1ml of 2.5% chlorpromazine per 3ml of 0.25% solution of novocaine. The dose is calculated by chlorpromazine. In addition to chlorpromazine, diazepam at a dose of 0.3mg/kg of body weight is used once a day to reduce cough attacks. In preschool children, diazepam (as well as salbutamol) can be given orally. For children 2-7 years salbutamol is administered 1-2mg 2-3 times a day, 8-14 years - 2mg three times a day. Most cough suppressants with whooping cough are ineffective. However, mucolytic agents are used to improve bronchial patency in whooping cough. It is more appropriate to administer aminophylline orally as a medicine in combination with potassium iodide having a distinct mucolytic effect.

In case of respiratory arrest **(apnea)** it is necessary to restore the airway patency as quickly as possible. The nose and mouth of the patient should be relieved of mucus and vomiting. Normal breathing is restored by rhythmic pressure on the chest and respirators. At frequent and prolonged apnea the child should be transferred to the intensive care unit, in the most severe cases - to artificial respiration. In addition, the introduction of diuretics (furosemide at a dose of 1-2mg/kg of body weight) is shown in such patients to normalize the water-electrolyte balance in the CNS. It is proved that the frequency and duration of apnea attacks with whooping cough reduces the introduction of

glucocorticoid hormones, especially hydrocortisone at a dose of 5-7mg/kg of body weight for 3-5 days. The dose of hormones is reduced gradually, as a rapid decrease in it can lead to the resumption of apnea and increased attacks of cough.

Prevention

In practice, the diagnosis of whooping cough is usually made up only in the stage of convulsive cough, respectively, delayed isolation of the patient, which, of course, reduces its epidemiological effectiveness. Therefore, the most important condition for successful implementation of anti-epidemic measures for whooping cough is early diagnosis. Isolation of the patient at home is carried out by carrying the patient to a separate room or behind the screen. Patients with severe and complicated forms of whooping cough are admitted to hospitalization, especially children under 2 years of age, ill children from families living in unfavorable living conditions, as well as from families with children under 6 months who have not had whooping cough. The isolation of the patient lasts until the 25th day from the beginning of the disease. The organization of the regime in the hospital requires special attention. It is necessary to ventilate the premises and disinfect the handkerchiefs, towels, dishes of the patient. Careful protection of patients from contact of a concomitant infection that is the cause of exacerbations and complications is required. For children under 7 years of age who have been in contact with patients who have not previously been ill and have not been vaccinated against whooping cough, they are quarantined for up to 14 days from the time of isolation of the patient. If the patient was not isolated and communication with him continued throughout the period of the disease, quarantine is imposed until the end of the infectious period in the patient. Due to the low resistance, the pathogen dies quickly, so there is no need for complete final disinfection after isolation of the patient. The focus of the infection is under medical supervision. When suspected whooping cough carry out bacteriological examination. In contact unvaccinated children, it is advisable to carry out chemoprophylaxis of

whooping cough erythromycin at a dose of 50mg/kg of body weight per day for 10-14 days. In order to prevent whooping cough erythromycin prescribing is indicated:

- for all patients in the first 3 weeks from the onset of the disease to reduce the intensity of the release of whooping cough in the environment;
- newborn babies born of mothers with whooping cough;
- children with chronic diseases of the bronchopulmonary system or heart, irrespective of vaccine history;
- pregnant women who have whooping cough within 3 days before and 10 days after birth. For the purpose of active immunization in Ukraine it is preferable to use whole-cell pertussis vaccine - a suspension of the first stage of pertussis germs, neutralized with formalin or merthiolate. This drug is used in association with diphtheria and tetanus toxoids (pertussis-diphtheria-tetanus, or DPT, vaccine). It is known that the DPT vaccine is the most reactogenic due to the whole-cell whooping cough component. A new generation vaccine with acellular whooping cough component - DPT (adsorbed acellular vaccine for the prevention of diphtheria, tetanus and whooping cough) was created to address this deficiency. DPT contains only three purified v antigens (whooping cough toxoid, flamentous hemagglutinin, and outer membrane protein, pertactin). Diphtheria and tetanus toxoids and cell-free pertussis vaccine components were adsorbed on aluminum salts. The vaccine is made in saline solution, as a preservative, it contains 2-phenoxyethanol (unlike other DPT where mercury salts are used as a preservative). According to the National Vaccination Calendar in Ukraine, acellular whooping cough vaccine is used for further vaccinations for children who have had post-vaccination complications for pre-vaccination DPT, as well as for all vaccinations for children at high risk of developing post-vaccination complications, especially those who have pathogens CNS. The whooping cough vaccination for children of 18 months of age in Ukraine is also carried out with the DPT vaccine.

Control questions:

- What age is the most susceptible for whooping cough?
- What is the pathogenesis of whooping cough?
- With what diseases do we carry out the diagnosis of pertussis?
- What first aid should you provide with apnea?
- How is whooping cough prevention performed in Ukraine.

CONVULSIVE SYNDROME. URGENT THERAPY.

Convulsions are a sudden onset of muscle contractions, accompanied by loss of consciousness and hypoxia of the brain. There are: clonic, tonic and mixed convulsions. The prevalence is localized and generalized; depending on frequency - episodic, serial and convulsive status. According to the mechanism of development, convulsive paroxysms are divided into convulsive reaction, convulsive syndrome and epileptic disease.

The convulsive reaction occurs in response to various stimuli caused by infection, intoxication, hypoxia. Most often, it occurs in infancy with increased convulsive readiness, which is due to the low differentiation of the cerebral hemisphere, against the background of the prevailing tone of the ascending reticular formation, the tone of the palliary system and high activity of the hippocampus under the influence of minor exogenous and endogenous influences. An example of a convulsive reaction is: hyperthermia, hypoglycemia, hypochloremia, hypocalcemia, hypomagnesemia, alkalosis, toxic effects of infection, overheating, severe mental trauma, poisoning.

The convulsive syndrome arises in the course of active processes in the nervous system and represents the extreme degree of the central excitation which goes beyond the standard.

Causes: mechanical traumas of the skull, intracranial abnormalities of the development of the skull bones (microcrania, craniosynostosis, internal hyperostosis), congenital brain defects (anencephaly, microcephaly, hydrocephalus, micro- and macrogyria), anomalies of the vessels of the kidney, toxoplasmosis, syphilis, intrauterine red embryopathy, cytomegalovirus encephalopathy), tumors, hereditary degenerative CNS lesions (diffuse brain sclerosis, amaurotic idiocy, atrophy). The convulsive syndrome is characterized by a recurrence of seizures.

Seizures in epilepsy occur on the background of a hereditary condition.

The onset of clonic-tonic seizures begins suddenly, there are signs of motor excitation, the child loses the contact with the environment, the gaze wanders, the eyeballs are fixed upwards, and then to the side, the head is thrown back, the trunk is fixed, the arms are bent at the joints, legs extend, the breathing stops, the pulse slows down, the skin becomes cyanotic. This can continue for one minute, after which the baby takes a deep breath. Clonic convulsions begin with facial twitching, then contractions occur in the extremities. Breathing becomes noisy, cyanosis decreases, the baby becomes pale, the heart rate accelerates. After the attack, the patient falls asleep.

In children, febrile seizures most commonly occur in 8-10%, with 50% recurring in the future. *Respiratory-effective seizures* are observed in infants with increased nervous excitability at the age of 7 months to 2 years. Such convulsions are provoked by fear, pain, forced feeding.

Meningoencephalic response is the most common cause of convulsions of early age that accompany infection, pneumonia, sepsis. In the pathogenesis, the main role is played by disorders of microcirculation, hypoxia and edema-swelling of the brain. There is a violation of biological oxidation in the Krebs cycle, lactate acidosis inhibits the processes of glycolysis, which leads to severe energy deficiency, impaired mineralization of neuroglia, hypernatremia. Clinically noted sharp excitation, single vomiting, positive meningeal signs, clonic convulsions, oriental strabismus, nystagmus, disturbance of consciousness, hypotension, arrhythmia of breathing, depression of respiratory and cardiovascular centers. Suppression of consciousness, lack of response to external stimuli, loss of sensitivity these are prognostically unfavorable signs.

First aid in seizures

Introduce ant-seizures medications (1-2drugs):

- phenobarbital 0.2% rn 1-5mg/kg per os to 2-3 times a day;
- benzodiazepines 0.2-0.3 mg/kg or 0.1ml per 1 year of life;
- GHB 20% 50-100mg/kg I/V slowly;

• Thiopental 10% 10mg/kg I/O slowly - single dose can be repeated after 3 hours, daily dose - 80mg/kg or 0.8ml/kg;

• metazolam 0.1mg/kg I/O slow single dose.

• Dehydration therapy: lasix (furosemide) intramuscular, intravenous at a dose of 1 - 3mg/kg/ day 1-2 times; magnesium sulfate at a dose of 0.2ml of 25% solution per 1 kg of body weight.

• Calcium preparations: calcium gluconate at a dose of 2-2.5 mg/kg.

• According to the indications - hydrocortisone 5-10mg/kg/ day 3 times; prednisone at a dose of 2-5mg/kg/ day.

Success against convulsive therapy can be achieved when we include measures to normalize gas exchange, eliminate dehydration and hypernatremia, **prevent and treat brain edema.**

Control questions:

• What can cause childhood seizures?

• What types of seizures do you know?

• What assistance do we provide for convulsive syndrome?

Calendar Of Preventive Vaccinations In Ukraine 18.05.2018 №947

Table 12

Age	Vaccination aginst					
1st day		Hepatitis B				
3-5 day	Tuberculosis					
1st month		Hepatitis B				
2 months			Diphteria, whooping cough, tetanus	Polyomielitis IPV	Hemophilic infection	
Age	**Vaccination aginst**					
4 months			Diphteria, whooping cough, tetanus	Polyomielitis IPV	Hemophilic infection	
6 months		Hepatitis B	Diphteria, tetanus	Polyomielitis OPV	Hemophilic infection	
12months						Measles, rubella, parotitis
18 months			Diphteria, whooping cough, tetanus	Polyomielitis OPV	Hemophilic infection	
6 years			Diphteria, tetanus	Polyomielitis OPV		Measles, rubella, parotitis
14 years			Diphteria, tetanus	Polyomielitis OPV		
18 years			Diphteria, tetanus			
Adults			Diphteria, tetanus			

Post-vaccination reactions and complications

- Temperature rise to 39 ^{0}C.
- Temperature rise above 39 ^{0}C (strong general).
- Temperature not recorded in medical records.
- Pain, soft tissue swelling> 50mm, hyperemia at the injection site > 80mm, infiltrate> 20mm (strong local).
- Lymphadenopathy.
- Headache.
- Irritability, sleep disorders.
- Rash of non-allergic genesis.
- Anorexia, nausea, abdominal pain, dyspepsia, diarrhea.
- Cataracts.
- Myalgia, arthralgia.
- Abscesses.
- Anaphylactic shock and anaphylactoid reactions.
- Allergic reactions (Quincke swelling, hives, Stevens-Johnson syndrome, Lyell).
- Hypotensive-hyporesponsive syndrome (acute cardiovascular failure, hypotension, decreased muscle tone, short-term disturbance or loss of consciousness, history of vascular disorders).
- Arthritis.
- Continuous high-pitched scream (3 hours or more).
- Febrile seizures.
- Afebrile convulsions.
- Meningitis / encephalitis.
- Anesthesia / Paresthesia.
- Acute paralysis.
- Vaccine-associated paralytic polio.
- Guillain-Barre syndrome (polyradiculoneuritis).

- Subacute sclerosing panencephalitis.
- Mumps, orchitis.
- Thrombocytopenia.
- Subcutaneous cold abscess.
- Superficial ulcer more than 10mm.
- Regional lymphadenitis (s).
- Keloid scar.
- Generalized BCG infection, osteomyelitis, osteitis.

Terms of development of post-vaccination reactions and complications:

BCG Vaccines. - local reaction, infiltrate 5-10mm h-4-6 t after vaccination, strong local reaction: swelling of soft tissues more than 20 mm, infiltrate more than 10 mm 5-7 days after revaccination. General reaction - fever without marked disruption of the general condition of the child, the first 2 days. Complications: Subcutaneous cold abscesses (violation of the technique of introduction). Superficial ulcer (violation of the technique of introduction). Regional lymphadenitis, colloid scar, osteomyelitis, generalized BCG - infection.

DTP, DT Local reaction - hyperemia of the skin, infiltrate. **Strong reaction** - soft tissue swelling more than 5 cm in diameter, infiltrate more than 2 cm. **General reaction**: fever without marked disturbance of the general condition of the child. **Strong reaction**: temperature rise above 38,6 ^{0}C. The first two days. **Allergic complications**: Quincke swelling, hives, exacerbation of allergic diseases (up to 10 days). In some cases, anaphylactic shock type reactions (up to 24 hours). **Colaptoid status** - decrease in muscle tone, sharp paleness, dizziness, drowsiness, cardiovascular or respiratory failure (7 days). Encephalopathy - impaired brain function, increased intracranial pressure (in children a continuous high pitch cry), impaired consciousness, convulsions, pathological reflexes, paresis. In some cases, encephalitis (up to 7 days).

Residual condition - occurrence of a seizure at a temperature less than 39 ^{0}C, acute nephritis, acute myocarditis, serous meningitis (up to 30 days).

Live polio vaccine Reaction - Intestinal dysfunction within 2-3 days. Complications - an impaired paresis of the extremities (often the lower) (7- 30 days), rash, swelling (up to 10 days).

Vaccination against rubella. Local reaction - hyperemia (1-3 days) **General reaction** - fever, rash, enlargement of lymph nodes (7-14 days, duration not more than 1-2 days). **Complications**: Hives (10 days) anaphylactic shock (24 hours). **Encephalopathy:** Convulsions associated with fever. Acute arthralgia, acute arthritis (14-30 days), transient polyneuropathy (14-30 days).

Hepatitis B vaccine. Local: pain, hyperemia, swelling, itching, redness, hardening of the injection site (1-4 days). General: short-term deterioration of руфдер, subfebrile temperature (1-3 days). **Complications:** Quince type edema, a reaction of a type of serum sickness with the development of arthritis and skin manifestations (erythema, ecchymosis, nodular erythema (up to 30 days). Hyena-Barre, optic neuritis (up to 30 days).

LCD. Local: hyperemia and tissue edema (1-3 days). General: fever 38 C, catarrhal phenomena from the upper respiratory tract and eyes, decreased appetite, nausea, headache, rarely - rash. (6-12 days). **Complications**: Allergic complications, polymorphic rash, Quincke swelling, arthralgia (12 days), anaphylactic shock (24 hours). **Encephalopathy:** convulsions, disturbance of consciousness, increase of intracranial pressure, in some cases encephalitis (10-15 days). **Toxic complications:** temperature above 38.6 ^{0}C, manifestations of intoxication, severe catarrhal symptoms, rash (6-16 days). Acute myocarditis.

LCD. Local: hyperemia (1-3 days). General: fever up to 37,5 ^{0}C, slight hyperemia of the throat, runny nose, decreased appetite, abdominal pain (4-10 days). Painless enlargement of the salivary glands. **Complications:** ршмуы, Quincke's swelling (1-16 days), ыушягкуы, disturbance of consciousness (7-15 days), fever (more than 38,5), vomiting, abdominal pain. Serious meningitis on a backround of fever.

Therapy of post-vaccination complications is performed by reviewing their nature, severity of the course, age of the child, and its individual features. The outcome of the disease depends on the timeliness and correctness of indications of therapeutic measures, especially in severe forms of complications. Complex therapy of post-vaccination complications carries out both specific (etiotropic) and nonspecific treatment (pathogenetic). Important place in the treatment of these patients occupies the right regimen, a rational diet and good care. Vaccinations are carried out in pediatric clinics only after the examination by the district pediatrician and after his / her appointment, because it is the district pediatrician who constantly monitors the child and only he / she can give a correct assessment of his / her health status, as well as perform measures before the vaccination of weak and often ill children.

If all vaccinations are given in accordance with the rules and the cold chain of vaccines storage is not compromised, a very low percentage is observed - 1 per 10,000 children, mainly in the form of local and general reactions.

Anaphylactic shock

The introduction of foreign protein into the body of a child can lead to the development of post-vaccination complications in the body of the child, which often occur against the background of hereditary and constitutional predisposition to allergic diseases One of the most severe manifestations of allergies is anaphylactic shock.

Clinical manifestations: discomfort, heat, shortness of breath, severe abdominal pain, inability to speak, weakness, dizziness, sharp pallor, fever, itching of the skin, mucous membranes, chest pain, in the region of the heart, hypoxia, hypoxemia, loss of consciousness, cardiovascular, respiratory, adrenal insufficiency, death is possible

Emergency aid:

- Interrupt further entry of the allergen into the body;
- When administering an allergen intramuscularly, the rubber band should be proximal to the site of administration for 20-30 minutes;

• Site of the injection must be punctured with a 0.1% 0.3-0.5ml solution of adrenaline hydrochloride or a 1% solution of mesatonone diluted in 5 ml saline;

• Urgent hospitalization in a ward or intensive care unit. The baby is transported in position on the side to prevent aspiration of the vomiting masses. Carry out oxygen therapy;

• 0.2-0.5ml of 1% solution of adrenaline hydrochloride are injected every 15 minutes under condition of shock.

• Intravenously 10% calcium chloride 1 ml per year of life is injected;

• Intravenously - antihistamines: 1% solution of dimedrol (0.5-3.0ml), suprastin - under 1 year of life 5mg, older children - 10mg; or 2.5% solution of pipolfen 0.1 ml per year of life;

• hormone therapy - 2.5% hydrocortisone solution (fast acting) 1.0ml for children under 1 year, 2ml – under 5, 3.0ml - under 10, 4.0ml - under 15;

• cardiac glycosides are administered in saline solution: 0.05% strophanthin 0.05 ml under 1 year of life, 0.1-0.2ml - under 5 years, 0.3 ml - for children older than 10 years; 0.06% of corglycone 0.1ml for children under 1 year, 0.2-0.5ml for children under 5 years, 0.6ml for children under 10 years, 0.75ml for children under 15 years;

• in case of the phenomena of bronchospasm 2.4% euphiline solution 0.5ml - under a year of life, 1.0-3.0ml - 2-5 years. life, 4.0ml - 6-10 years, 5.0ml - 11-15 years of life;

• in cases of bronchospasm urgent intubation or tracheotomy, mechanical ventilation;

• with arterial hypotension, a 0.2% solution of norepinephrine is injected interavenous 0.1ml for children under 1 year, 0.2-0.3ml for children 2-5 years, 0.4-0.5ml for children 6-10 years, 0, 6-0,8ml for children 11-15 years. life;

• CVT normalization is achieved by increasing the OCC through the introduction of plasma substitutes;

- infusions are carried out through the central veins - subclavian or internal jugular.

The effectiveness of therapy depends on the timing of the started care, the presence of a anti-shock first-aid kit at a health care provider. In order to prevent anaphylactic shock, the doctor should always collect the allergic history comprehensively from the parents and note in the medical records, conduct allergic tests before the introduction of antibiotics, foreign sera and gamma globulins, in cases of confusion perform antihistamine protection.

Control questions:

- What are possible post-vaccination complications after vaccination?
- What is anaphylactic shock?
- What is the urgent care for anaphylactic shock?

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TESTS. SITUATIONAL TASKS

1 And that applies to the first line of defense against infectious agents:
Undamaged skin

Phagocytosis

Activation of the complement

Production of antibodies

Antigen recognition

2 Main cellular component specific immunity :

Lymphocytes

Mast cells

Neutrophils

Macrophages

Basophils

3 Basic components of humoral specific immunity:

Antibodies

Complement

Lysozyme

Interferon

Lymphokines

4 Complement system – is:

The system enzymes

The system cells

The system antibodies

5 All these are the result of complement activation, except:

Reduced activity of phagocytosis

Expansion vessels and increase their permeability

Production of inflammatory mediators

Cytolysis or hemolysis

Opsonization

6 Complement component deficiency which often occurs during recurrentmeningococcus infection:

C5

C2

C3

C6

7 What type of white blood cells takes major part in phagocytosis?

Neutrophils

Eosinophils

Basophils

Monocytes

Mast cells

8 What mononuclear cells belonging to the phagocytic system?

Monocytes, macrophages and promonocytes

Monocytes and promonocytes

Monocytes and macrophages

Lymphocytes and monocytes

9 What is the matter with antiviral activity produced by macrophages?

Interferon

Lysozyme

Fibrinogen

Prostaglandin

10 What protective monocytes and perform macrophages?

All of the right

The presentation of antigen lymphocytes

Phagocitosis

Induction of immune response

Secretion of biologically active molecules

11 Which of the acute phase proteins is the most sensitive and rapidly responsive indicator of acute inflammation?

C-reactive protein

Complement

Ceruloplasmin

Haptoglobin

Fibrinogen

12 Indicate classes of immunoglobulins, which appears first in the immune response?

Ig M

IgA

Ig E

Ig G

13 What class of antibodies is dominant inmucosal secretions and prevent atattachment of microorganisms to epithelialcells?

Ig A

Ig G

IgM

Ig E

Ig D

14 Which classes of immunoglobulins capable oflook into the placenta?

Ig G

Ig A

Ig M

Ig E

Ig D

15 That applies to the central lymphoidof a person?

Bone and thymus lobes

Lymph nodes and thymus

Lymph nodes and spleen

Lymph nodes and plaques peyerovi

16 By the function of T lymphocytes does not apply?

Synthesis antibodies

Participation in delayed hypersensitivity reactions

Participation in the reactions cytolysis

Regulation of the immune response

Protection against intracellular pathogens

17What helper cells have on their surface?

Molecules CD 4 +

Molecules CD 8+

Molecules Ig M

Molecules Ig D

18 What correctly describes the humoralimmune response?

The result is the production of antibodies differentiated B-lymphocytes

Part of innate immunity

It involved only T cells

For the presentation of antigens B-lymphocytes need macrophages

19 The main clinical manifestations of B-cell failure concerns?

Increased susceptibility to bacterial infections

Impaired phagocytosis

Reduced complement

Increased sensitivity to parasitic infections

Lymphadenopathy

20 In addition to antiviral activity, interferonsmay affect other functions of cells. The introduction of exogenous interferon affects not?

Attaching antibodies to oats

Cell growth

The activity of natural killers cells

Strengthening the function of macrophages

Increased expression of cell surface antigens (HLA)

21 for vaccination against diphtheria use?

Toxoid

Killed goals microorganisms

Components of microorganisms

Living virulent strains

Live attenuated strains

22 For vaccination against Haemophilus influenzae type b infection using?

Intramuscular polysaccharide complex with a protein carrier

Intramuscular injection of killed microorganisms

Intramuscular toxoid

Peroralnyj reception of weakened live virus

Subcutaneous administration of attenuated live virus

23 For vaccination against rubella using?

Subcutaneous administration of attenuated live virus

Intramuscular injection of killed microorganisms

Intramuscular toxoid

Intramuscular polysaccharide complex with a protein carrier

Peroralnyj reception of weakened live virus

24 Two hours after enter of the DTP vaccine to 4-month boy's temperature increase to 38 ^ C, anxiety. What will doctors tactic during examination of the patient at the age of 5 months?

Introduce DTP vaccine, recommending preventive treatment to prevent hyperthermia reaction

Delay vaccination against pertussis and enter tetanus and diphtheria toxoid;

Delay vaccination against pertussis and enter diphtheria and tetanus toxoids a low dose (ADP-M)

Enter half of the usual dose of DTP vaccine

Delay vaccination until the age of 12 months

25 Given statements are valid for atopic dermatitis (eczema), except:

Vaccination is contraindicated with eczema

The characteristic itching

The rash is localized on flexor surfaces

Family anamnesis is common asthma and hay fever

The manifestations occur in early childhood

26 Vaccination with genetically-engineered vaccine with surface antigen of hepatitis B virus by three times intramuscular enter can prevent disease listed, except:

Dropsygallbladder

Cirrhosis

Hepatocellular carcinoma

Acute fulminant hepatitis

27 Which resulted from groups not recommended flu shots:

Children up to 6 months

Medical workers

Patients continued taking aspirin

Patients with diabetes

Family members, who are concerned about the risk to the patient

28 A child positive Mantoux test just all of the right statements, except:

It shows that the child is contagious

It appears within 2-10 weeks after infection

It is an indication for starting antibiotic therapy

It can be a negative for a short time after immunization with live agent or live virus

It may indicate infection with atypical mycobacteria

29 For the routine vaccinations to children, the vaccine used, except:

Pneumococcal

BCG

Mumps vaccine

Against Hepatitis B

Ternary vaccine against mumps, rubella, measles

30 How to proceed with further grafting healthy baby 10 months, which two months ago suffered pneumonia?

Continue vaccination, do DTP vaccination the third time

Assume that prior vaccination has lost action, again to start ternary DTP vaccination

Consider two vaccinations sufficient 1-1,5 year do DPT revaccination

Continue vaccination with DTA-M toxoid

31 On the 10th day after vaccination DPT vaccine, a childs temperature was increased to 37.5 C, rhinorrhea, coughing. Most likely it can be regarded as:

The begining of respiratory infection

The normal course of post-vaccination process

Contavention of vaccination procedure

Complications to the vaccine

32On the 2nd day after vaccination with DPT vaccine observed rising of temperature to 37.5 C at the injection site - slightly painful consolidation. It is regarded as:

Normal flow of vaccine process

Contravention of vaccination procedure

Hypersensitivity to the vaccine, in the future must refuse of vaccination

The reaction to the shot

33Which of vaccinations drug 4-year old child with atopic dermatitis (eczema), which had not previously been immunized by DPT vaccine:

ADP toxoid

ADP-M toxoid

administrative toxoid

34 Who is not indicated for routine vaccination againstmeasles, mumps and rubella if children earlier werenot suffered from these infections and were not vaccinated.

The child is 1 year and 1 month win hemoglobin level 78 g/l

The child is 1 year and 2 months with 2nd degree thymomegaly

The child is 1 year with disbacteriosis

The child is 1 year and 2 months. That first year of life were 6 times

Infected by URTI (Upper respiratory tract infection)

The child is 1 year that HIV positive.

35 At the day of vaccination against mump, measles and pertussis the childs temperature increased to 38.2 C, rhinorrhea, cough. What you need to do?

Prorogue vaccination until temperature normalization and convalescence

Enter vaccine against measles

Cancel vaccintaion, consider the question about vaccination after motnh of convalescence

Enter vaccine against measles under imunoglobulin protection

36HIV-infected children can not be vaccinated with:

Living polio vaccine

DPT vaccine

Measles vaccine

Flu vaccine

Vaccine of Naemophilusinfluenze b infection

Pneumococcal vaccine

37 **Which of these children can not vaccinated against tuberculosis in the hospital?**

Premature baby (body weight 1900) on the 8th day of life

Healthy child (body weight 3800h.)

On the 5th day of lifePremature baby (body weight 2300h.)

On the 3rd day of life

All answers are correct

38 Have formed a complete post-vaccination immunity in a child if 7 days after vaccination with live measles vaccinenormal human immunoglobulinwas entered to her?

No

Yes

39Immediate vaccination at stake is not made against:

Mumps

Diphtheria

Polio

Measles

40 **Post-vaccination lesions of the nervous system include:**

All answers are correct

Post-vaccination encephalitis

Meningoencefalitis

Encephalopathy

TASK

1.On the 21 day after appearance of vesiculous chickenpox rash a 7-year-old child developed ataxia, nystagmus, intention tremor, muscle hypotonia. Liquor analysis shows a low-grade lymphocytic pleocytosis, slightly increased protein rate. What complication is it?

Encephalitis

Pneumonitis

Purulent meningitis

Acute nephritis

Postherpetic neuralgia

2.A child is 9 months old. The patients body temperature is 36,7°C, the skin is pale, humid, there is pain in leg muscles. There is no extremities mobility, sensitivity is present. The child has been diagnosed with poliomyelitis. The causative agent of this disease relates to the following family:

Picornavirus

Rotavirus

Adenovirus

Tohovirus

Paramyxovirus

3.A child is 2 years old. The child complains of hoarse voice, dyspnea with obstructed inspiration. The disease started 3 days ago from dry cough and nose stuffiness. Objectively: general condition is unbalanced, stridor is present. The childs skin is pale. Body temperature is 37,7°C. The palatine arches are hyperemic. There is no deposit. Heart sounds are rhythmic. Auscultation of lungs reveals rough breathing sounds, crepitation is absent. Parainfluenza virus has been detected in nasopharynx lavage. What is the most likely diagnosis?

Acute laryngotracheitis

Diphtheria

Epiglottitis

Foreign body

Laryngospasm

4.A 3-year-old child was playing in a playpen when he suddenly developed paroxysmal cough and shortness of breath. Objectively: dry cough, mixed dyspnea. Lung auscultation revealed some wheezes. Breathing sounds on the right are diminished. The child doesnt mix with other children. Immunization is age-appropriate. What pathological condition can be suspected?

Foreign body in the respiratory tracts

Acute respiratory viral infection

Bronchial asthma

Pneumonia

Pertussis

5.A 5-year-old child developed an acute disease starting from body temperature rise up to 38,5°C, running nose, cough and conjunctivitis. On the 4th day the child presented with maculo-papular rash on face. Body temparature rose again up to 39,2°C. Over the next few days the rash spread over the whole body and extremities. Mucous membrane of palate was hyperemic, there was whitish deposition on cheek mucous membrane next to molars. What is your provisional diagnosis?

Measles

Yersinia

Enterovirus diseases

Acute viral respiratory infection

Rubella

6.An 8-year-old boy fell ill acutely: he presents with fever, weakness, headache, abdominal pain, recurrent vomiting, then diarrhea and tenesmus. Stools occur 12 times daily, are scanty, contain a lot of mucus, pus, streaks of blood. His sigmoid gut is tender and hardened. What is your diagnosis?

Dysentery

Cholera

Staphylococcal gastroenteritis

Escherichiosis

Salmonellosis

7. A 4 month old child fell seriously ill: body temperature rose up to 38,5°C, the child became inert and had a single vomiting. 10 hours later there appeared rash over the buttocks and lower limbs in form of petechiae, spots and papules. Some haemorrhagic elements have necrosis in the centre. What is the most probable disease?

Meningococcemia

Influenza

Rubella

Haemorrhagic vasculitis

Scarlet fever

8.A 12 year old child has the ulcer disease of stomach. What is the etiology of this disease?

Intestinal bacillus

Lambliosis

Salmonella

Helicobacter pylory

Influenza

9.A 2 year old girl has been ill for 3 days. Today she has low grade fever, severe catarrhal presentations, slight maculopapular rash on her buttocks and enlarged occipital lymph nodes. What is your diagnosis?

Rubella

Measles

Scarlet fever

Pseudotuberculosis

Adenoviral infection

10.A 3 year old child has been suffering from fever, cough, coryza, conjunctivitis for 4 days. He has been taking sulfadimethoxine. Today it has fever up to 39 ^{0}C and maculopapular rash on its face. Except of rash the childs skin has no changes. What is your diagnosis?

Measles

Rubella

Allergic rash

Pseudotuberculosis

Scarlet fever

11.A 10 month old boy has been ill for 5 days after consumption of unboiled milk. Body temperature is 38-39C, there is vomiting, liquid stool. The child is pale and inert. His tongue is covered with white deposition. Heart sounds are muffled. Abdomen is swollen, there is borborygmus in the region of ubbilicus, liver is enlarged by 3 cm. Stool is liquid, dark-green, with admixtures of mucus, 5 times a day. What is the most probable diagnosis?

Salmonellosis

Staphylococcal enteric infection

Rotaviral infection

Acute shigellosis

Escherichiosis

12.A 3 year old child fell acutely ill, body temperature rose up to 39,5C, the child became inert, there appeared recurrent vomiting, headache. Examination revealed positive meningeal symptoms, after this lumbal puncture was performed. Spinal fluid is turbid, runs out under pressure, protein concentration is 1,8 g/l; Pandy reaction is +++, sugar concentration is 2,2 millimole/l, chloride concentration - 123 millimole/l, cytosis is 2,35×109 (80% of neutrophils, 20% of lymphocytes). What is the most probable diagnosis?

Purulent meningitis

Subarachnoid haemorrhage

Brain tumour

Serous viral meningitis

Serous tuberculous meningitis

13.A 7 year old girl has mild form of varicella. Headache, weakness, vertigo, tremor of her limbs, ataxia, then mental confusion appeared on the 5th day of illness. Meningeal signs are negative. Cerebrospinal fluid examination is normal. How can you explain these signs?

Encephalitis

Neurotoxic syndrome

Myelitis

Meningitis

Meningoencephalitis

14.A 3 m.o. child fell seriously ill, body temperature rised up to 37,8° there is semicough. On the 3-rd day the cough grew worse, dyspnea appeared. On percussion: tympanic sound above lungs, on auscultation: a lot of fine moist and wheezing rales during expiration. What is the most probable diagnosis?

Acute respiratory viral infection, bronchiolitis

Acute respiratory viral infection, bronchitis

Acute respiratory viral infection, bronchopneumonia

Acute respiratory viral infection, bronchitis with asthmatic component

Acute respiratory viral infection, focal pneumonia

15.A 7 y.o. girl fell ill abruptly: fever, headache, severe sore throat, vomiting. Minute bright red rash appear in her reddened skin in 3 hours. It is more intensive in axillae and groin. Mucous membrane of oropharynx is hyperemic. Greyish patches is on the tonsills. Submaxillary lymph nodes are enlarged and painful. What is your diagnosis?

Scarlet fever

Enteroviral infection

Rubella

Pseudotuberculosis

Measles

16.A 7-year-old child is sick for 2 weeks with running nose, was taking nasal drops. The boy suffers with alimentary allergy. He applied to doctor due to suppurative and bloody discharges from nose, maceration of ala nasi and upper lip. Rhinoscopy results: there are whitish-greyish areas at nasal septum. Mucous membrane of oropharynx is not changed. What is the most probable disease?

Diphtheria of the nose

Rhinovirus

Sinusitis (maxillar sinus))

Allergic rhinitis

Adenovirus

17.A child is 4 years old, has been ill for 5 days. There are complaints of cough, skin rash, to- 38,2°C, face puffiness, photophobia, conjunctivitis. Objectively: there is bright, maculo-papulous, in some areas confluent rash on the face, neck, upper chest. The pharynx is hyperemic. There are seropurulent discharges from the nose. Auscultation revealed dry rales in lungs. What is the most likely diagnosis?

Measles

Adenoviral infection

Rubella

Enterovirus exanthema

Scarlet fever

18.A 5-year-old child had strong headache, vomiting, ataxy, dormancy, discoordination of movements, tremor of the extremities on the 8th day of the disease. It was followed by rise in body temperature, vesiculosis rash mainly on the skin of the body and the hairy part of the head. At the second wave of the fever a diagnosis of encephalitis was given. What disease complicated encephalitis in this case?

Chicken pox

Enterovirus ifection

Herpetic infection

Measles

German measles

20.An 11-year-old girl has been immunized according to her age and in compliance with the calendar dates. What vaccinations should the children receive at this age?

Diphtheria and tetanus

Polio

Pertussis

Hepatitis

TB

21.A 6-year-old child complains of frequent liquid stool and vomiting. On the 2nd day of desease the child presented with inertness, temperature rise up to 38,2°C, Ps- 150 bpm, scaphoid abdomen, palpatory painful sigmoid colon, defecation 10 times a day with liquid, scarce stool with mucus and streaks of green. What is a provisional diagnosis?

Shigellosis

Salmonellosis

Yersiniosis

Escherichiosis

Intestinal amebiasis

22.A 1,5-year-old child fell ill acutely with high temperature 38oC, headache, fatigue. The temperature declined on the fifth day, muscular pain in the right leg occured in the morning, there were no movements and tendon reflexes, sensitivity was reserved. What is the initial diagnosis?

Polyomyelitis

Hip joint arthritis

Polyartropathy

Osteomyelitis

Viral encephilitis

23.A 10-year-old girl was admitted to a hospital with carditis presentations. It is known from the anamnesis that two weeks ago she had exacerbation of chronic tonsillitis. What is the most likely etiological factor in this case?

Streptococcus

Staphylococcus

Pneumococcus

Proteus

Klebsiella

24.An infant aged 1 year on the third day of common cold at night developed inspiratory stridor, hoarse voice and barking cough. Physical examination revealed suprasternal and intercostal chest retractions. There is a bluish skin discoloration moistly seen over the upper lip. The respiratory rate is 52 per min and pulse- 122 bpm. The body temperature is 37,5°. What disease does the infant have?

Acute infectious croup due to viral laryngotracheitis

Acute epiglottitis

Bronchopneumonia without complications

Acute bronchiolitis with respiratory distress

Acute laryngitis

25.A 7-year-old child is sick for 2 weeks with running nose, was taking nasal drops. The boy suffers with alimentary allergy. He applied to doctor due to suppurative and bloody discharges from nose, maceration of ala nasi and upper lip. Rhinoscopy results: there are whitish-greyish areas at nasal septum. Mucous membrane of oropharynx is not changed. What is the most probable disease?

Diphtheria of the nose

Rhinovirus

Sinusitis (maxillar sinus))

Allergic rhinitis

Adenovirus

26.A 5-year-old child had strong headache, vomiting, ataxy, dormancy, discoordination of movements, tremor of the extremities on the 8th day of the disease. It was followed by rise in body temperature, vesiculosis rash mainly on the skin of the body and the hairy part of the head. At the second wave of the fever a diagnosis of encephalitis was given. What disease complicated encephalitis in this case?

Chicken pox

Enterovirus ifection

Herpetic infection

Measles

German measles

27.An 8-year-old child was hospitalized for fever up to 39,8°C, inertness, moderate headache, vomiting. Examination revealed meningeal symptoms. Lumbar puncture was performed. The obtained fluid had raised opening pressure, it was transparent, with the cell count of 450 cells per 1mcL (mainly lymphocytes - 90%), glucose level of 2,6 mmol/l. What causative agent might have caused the disease in the child?

Enterovirus

Meningococcus

Staphylococcus

Pneumococcus

Kochs bacillus

28.A 3 year old child has been suffering from fever, cough, coryza, conjunctivitis for 4 days. He has been taking sulfadimethoxine. Today it has fever up to 39C and maculopapular rash on its face. Except of rash the childs skin has no changes. What is your diagnosis?

Measles

Rubella

Allergic rash

Pseudotuberculosis

Scarlet fever

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